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Field Guide to WILDLIFE DISEASES

General Field Procedures and Diseases of Migratory Birds

UNITED STATES DEPARTMENT OF THE INTERIOR
FISH AND WILDLIFE SERVICE
Resource Publication 167

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Field Guide to
WILDLIFE DISEASES

Volume 1. General Field Procedures and Diseases of Migratory Birds

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Field Guide to Wildlife Diseases

Contents

Page

Foreword v

Acknowledgments vi

Introduction vii

Section 1. General Field Procedures

1. Recording and Submitting Specimen History Data. *J. Christian Franson* . 1
2. Specimen Collection and Preservation. *J. Christian Franson* 7
3. Specimen Shipment. *J. Christian Franson* 13
4. Disease Control Operations. *Milton Friend and J. Christian Franson* . . . 21
5. Euthanasia. *Patricia A. Gullett* 59

Section 2. Diseases of Migratory Birds

Bacterial Diseases

6. Avian Cholera. *Milton Friend* 69
7. Avian Botulism. *Louis N. Locke and Milton Friend* 83
8. Avian Tuberculosis. *Thomas J. Roffe* 95
9. Avian Salmonellosis. *Richard K. Stroud and Milton Friend* 101
10. Chlamydiosis. *Louis N. Locke* 107

Viral Diseases

11. Duck Plague. *Christopher J. Brand* 117
12. Inclusion Body Disease of Cranes. *Douglas E. Docherty* 129
13. Avian Pox. *Wallace R. Hansen* 135

Fungal Diseases

14. Aspergillosis. *Louis N. Locke* 145

Parasitic Diseases

15. Sarcocystis. *Benjamin N. Tuggle* 153
16. Gizzard Worms. *Benjamin N. Tuggle* 159
17. Nasal Leeches. *Benjamin N. Tuggle* 165

Toxic Diseases

18. Lead Poisoning. *Milton Friend* 175
19. Oil Toxicosis. *Patricia A. Gullett* 191

Appendix A. Sample Specimen History Form 197
Appendix B. Sources of Wildlife Disease Diagnostic Assistance in the United States 199
Appendix C. Sources of Supplies Used in Collecting, Preserving, and Shipping Specimens 200
Appendix D. Mammalian Dissection Illustration and Information 202
Appendix E. Common and Scientific Names of Wildlife 205
Glossary of Technical Terms 209
Glossary of Animal Terms 213
Index 215

Dedication

To our colleagues in the wildlife disease field and to wildlife resource managers everywhere for their contributions and their dedication, and to Robert E. Putz and Richard N. Smith, colleagues and friends, whose administrative knowledge, perseverance, and support were fundamental to the establishment of the National Wildlife Health Center.

Foreword

Diseases of wildlife are not new; in fact, they have been recognized for centuries. More than 2,000 years ago references to diseases in wildlife were recorded in the Bible as well as in the writings of Homer and Aristotle. As a science, however, this subject has been slow in developing and in receiving appropriate professional recognition.

In 1954, 36 scientists who shared a common interest in diseases of wildlife gathered at the North American Wildlife and Natural Resources Conference in Washington, D.C., and established the Wildlife Disease Association. For the first time, professionals who had a common interest in diseases of wildlife were formally brought together. The growth and development of the Wildlife Disease Association in subsequent years illustrates an increasing interest in the subject, but it was a slow and laborious process that did not significantly change the status of disease as an ecologic factor in wildlife management.

In the early 1970's, the publication of a series of books by Davis et al., titled *Infectious Diseases of Wild Mammals* (1970), *Infectious and Parasitic Diseases of Wild Mammals* (1971), and *Parasitic Diseases of Wild Mammals* (1971), provided the first real references for scientists working with wildlife diseases. In addition, they served as the first "textbooks" for academia and were a catalyst for the development of wildlife disease courses in universities and colleges.

To the practicing wildlife manager or biologist, however, "wildlife disease" was still an unknown entity which received limited consideration in the management of wildlife populations unless large, spectacular die-offs occurred. Field personnel still had few tools and limited background with which to deal with diseases in wildlife.

Slowly but surely our knowledge and appreciation of diseases in wildlife have increased through veterinary and human medicine research, publications, and conferences. The wildlife disease practitioner benefited from these advances and the subject evolved as a profession.

This *Field Guide to Wildlife Diseases* is the first publication to provide wildlife field personnel with current scientific information about wildlife diseases in an easily understandable and eminently practical form. It provides a readable text supported by appropriate illustrations and photographs on how to collect, prepare, and submit wildlife specimens for postmortem examination. In addition, the Guide contains specific chapters on the major diseases of wild birds. These are written to help field personnel to understand and recognize these diseases and to provide an appropriate course of action involving the integration and utilization of the services of wildlife health specialists.

Wildlife managers and biologists are the key to what is happening in the field, be it in health surveillance, the detection of a disease problem, or disease control. To successfully fulfill this key role, the manager or biologist must understand and feel at ease with the subject matter. This manual provides that understanding and assurance.

This Guide provides the educator with a much needed academic tool—a laboratory manual for use in wildlife disease courses. This will strengthen wildlife disease education both on and off campus.

The National Wildlife Health Center and its staff have made a truly significant contribution to future wildlife populations with the preparation of this Guide. It is professionally done, scientifically excellent, appropriate to intended users, and much needed.

Daniel O. Trainer, Dean
College of Natural Resources
University of Wisconsin–Stevens Point

Acknowledgments

Special thanks to Myrna Burwitz for her efficiency, timeliness, and good cheer in typing the many revisions of this manuscript, often under pressure of deadlines, and to James Runningen for his extraordinary efforts in producing hundreds of quality color photographic prints needed for the manuscript review process.

We also thank Louis Locke, NWHC Training Officer, and Richard Stroud, NWHC Diagnostic Section Head from 1980 to 1987, for providing and explaining animal dissections from which a number of illustrations were prepared. Special thanks to Terry Amundson, Wisconsin Department of Natural Resources, for providing a deer carcass from which illustrations were made.

The photographs in this volume were taken by the following persons: Milton Friend, James Runningen, Ronald Windingstad, Wallace Hansen, Dave Trauger, Jerry L. Capps, Steve Schmidt, William Bair, and Carl Batha. Their respective photographs are identified by initials in italics after each caption.

Introduction

Most field biologists, refuge managers, and others who work with free-living animals encounter diseased wildlife at some time. These encounters may involve a few animals or large-scale die-offs, the need to seek assistance or initiate independent actions, and a need to respond to an inquiring public about the nature and seriousness of the problem. Frequently, such events provoke a personal interest in better understanding the cause of the disease.

This volume of the field guide provides practical information on selected diseases of free-living migratory birds and has been developed specifically to assist National Wildlife Refuge managers and other users of the National Wildlife Health Center. It is intended as a practical guide to help field biologists deal with disease problems whether they act independently or interact with wildlife disease specialists and personnel of animal disease diagnostic laboratories.

Our approach to developing this publication has been as much as possible to relate information to field situations, to use plain language rather than technical terms, and to illustrate as much of the information as possible.

Section 1 contains general information applicable to nearly all disease problems of wildlife. Section 2 contains individual chapters on specific diseases of migratory birds. We have made no attempt to cover all diseases of North American wildlife, as this guide is not intended as a textbook. Rather, the diseases we have included meet two criteria: (1) the disease condition is of great importance or frequently encountered, and (2) field signs or pathology associated with the condition can be illustrated in a manner that will help field managers to recognize the disease.

We have defined disease in the broad sense and have not limited this guide only to conditions caused by infectious agents. Rather, we have included diseases caused by viruses, bacteria, fungi, parasites, toxins, and mechanical processes (oiling). Similarly, the term "animal" is considered in a broad sense and includes all vertebrate species, not just mammals. Supplemental readings have been selected for their value in enhancing understanding of specific diseases and for their relative availability.

Our format for the individual disease chapters addresses specific questions commonly asked by field biologists and the public. These questions include the common names of each disease (*Synonyms*), what caused the disease (*Cause*), relative susceptibility of various animals (*Species Affected*), where the disease is most common as well as general occurrence (*Distribution*), when the disease is most likely to occur (*Seasonality*), how to recognize the disease (*Field Signs*), what to look for when examining carcasses (*Gross Lesions*), what specimens to collect and how to preserve them for examination by disease diagnostic laboratories (*Diagnosis*), what can be done to combat each disease (*Control*), and hazards to man associated with handling diseased animals (*Human Health Considerations*).

This field guide is written to be relevant, easily understood, and sufficiently detailed to help individuals in remote locations, as well as those who have more ready access to disease control specialists and disease diagnostic laboratories. However, information contained within this publication will be most useful when combined with some training and experience involving diseases of wildlife. We urge readers to expand the utility of the information provided by consulting with disease specialists and diagnostic laboratories whenever possible.

We invite and welcome readers to comment on ways future chapters can be improved. Comments should be addressed to the Director, National Wildlife Health Center.

Milton Friend, Director
National Wildlife Health Center

Section 1.

General Field Procedures

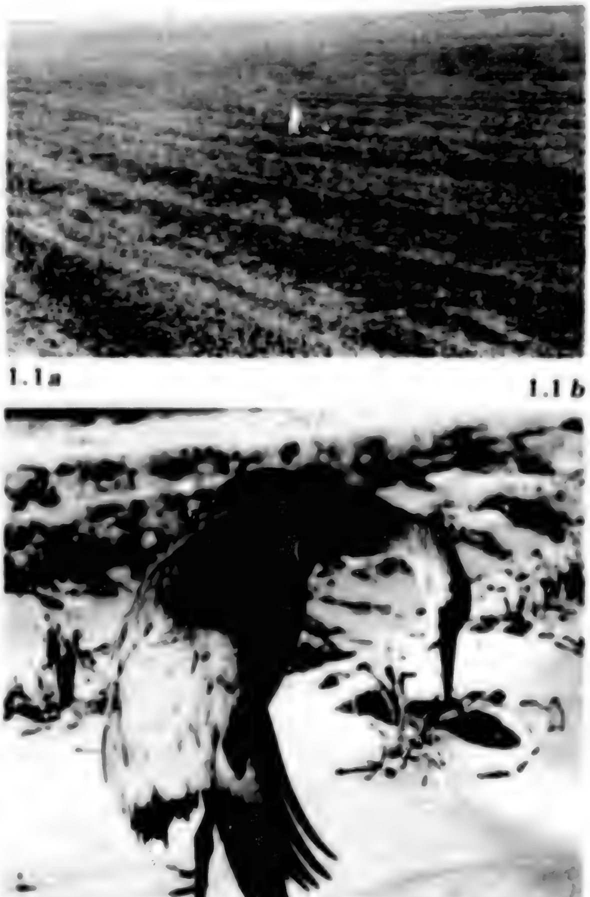
Chapter 1.
Recording and Submitting Specimen History Data

J. Christian Franson

Webster defines history as "a chronological record of significant events." In wildlife disease investigations, determining the history or background of a problem is the first significant step in establishing a diagnosis. You can greatly assist the diagnostic process by providing a thorough history with specimens you submit. This information is also of value in understanding the natural history of disease outbreaks, and is difficult if not impossible to obtain after the event has occurred. Detailed field observations during the course of a die-off and investigation of significant events preceding it also provide valuable information on which to base corrective actions. Remember, the most helpful information is that which is obtained at the time of the event by a sensitive and aware observer.

What Information Should Be Collected

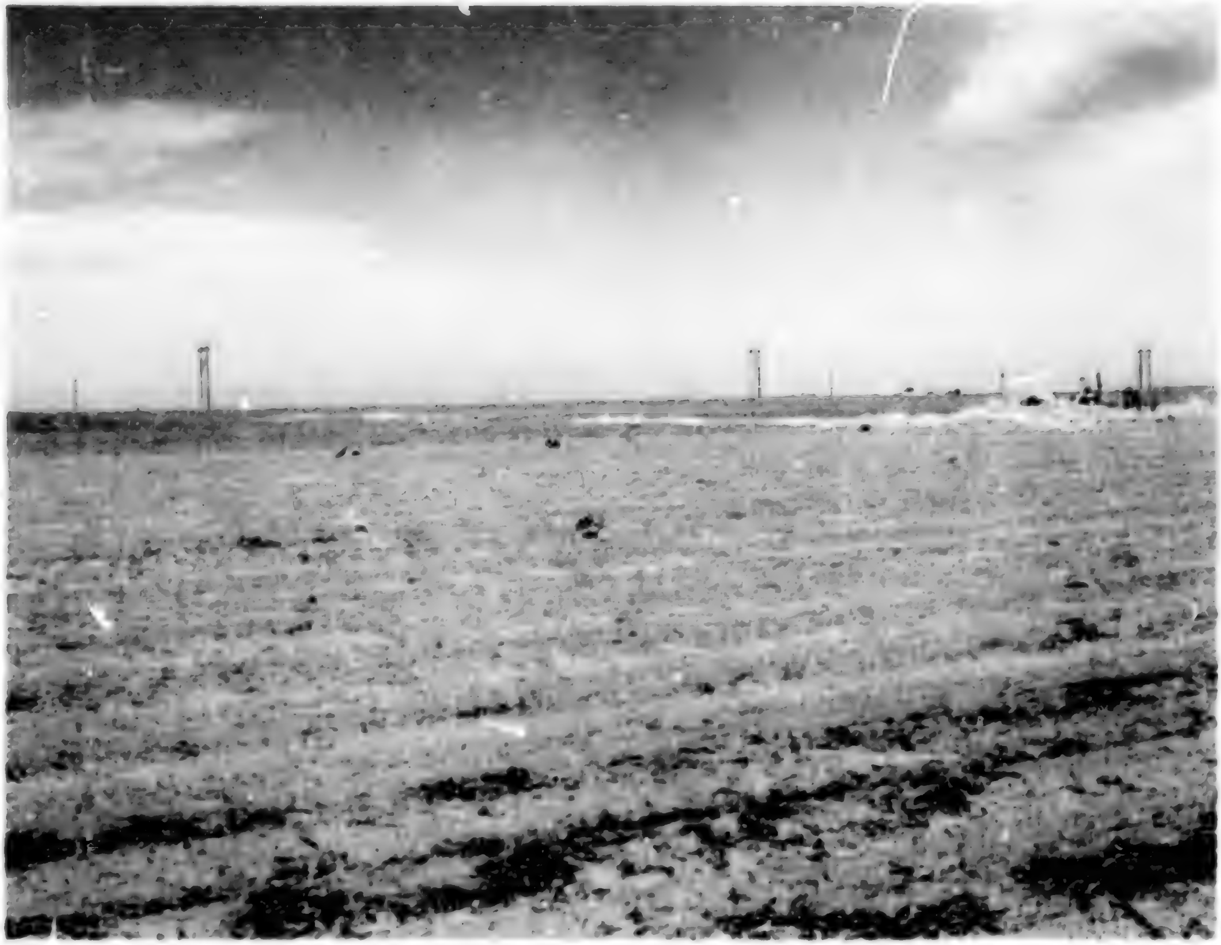
The rule here is, "If in doubt, write it down." Avoid preconceptions that limit the information collected. A sample specimen history form is provided in Appendix A. Be as complete as possible; what seems irrelevant in the field may be the key to a diagnosis. Photographs are sometimes helpful, but do not delay sending specimens or a written history until the photographs are available. Indicate that photographs have been taken and will be forwarded when available. Photographs should focus on specific information you wish to convey such as environmental conditions and the appearance of sick animals or lesions. A common error is to photograph from too great a distance so that insufficient subject detail is present (Fig. 1.1). Fill the viewing screen with as much of a single subject as possible while maintaining sharp focus and including sufficient other material to provide a frame of reference for size, habitat, orientation, or other factors (Fig. 1.2).



1.1a

1.1b

Fig. 1.1. Examples of (*a*) poor and (*b*) good use of photography to record information associated with wildlife disease problems. Distant photo of sick bird in field does not provide sufficient detail to convey useful information. Closeup shot of sick bird clearly illustrates the species, shows the wing and neck droop, and shows that breast muscles are not wasted. *RW*



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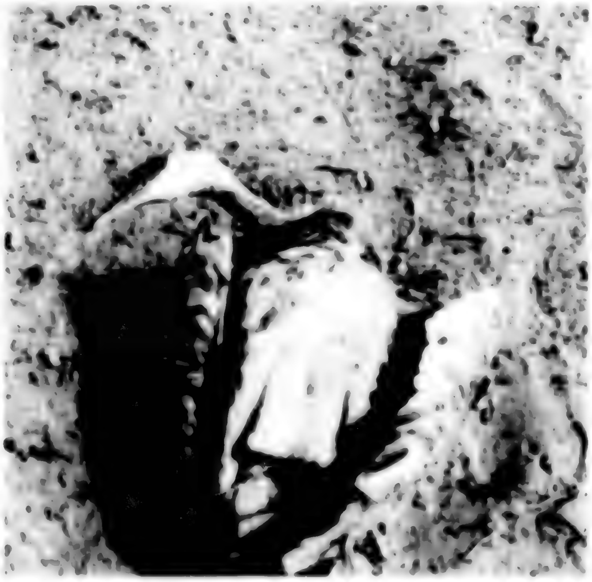


Fig. 1.2. Examples of poor and good photography to record environmental conditions associated with wildlife disease problems. *(a)* Distant shot of field of peanuts with dead birds does not clearly show birds. *(b)* Closeup shot clearly shows both birds and peanuts. *R.W.*

The following basic information is helpful in diagnosing the cause and assessing the severity of a wildlife health problem.

Environmental Factors

Determine if the start of mortality coincided with any unusual event. Environmental changes such as storms, precipitation, and abrupt change in temperature are potential sources of stress that can contribute to disease outbreaks. A food shortage may degrade the condition of animals and increase their susceptibility to disease. Changes in water levels on an area may concentrate or disperse birds, alter the accessibility of spent lead shot, or cause an invertebrate die-off that could lead to an avian botulism problem.

The quality of water used for impoundments may be significant; poor water quality may contribute to avian botulism or could be a primary factor in mortality if contamination is severe. Recent pesticide applications and other habitat or crop management practices should also be recorded, as should previous disease problems in the area.

Estimating Disease Onset

When estimating the onset of disease, consider (1) the relation between on-site activities that could have resulted in the detection of sick or dead animals if they were present and the date diseased animals were first observed, and (2) the percentage of carcasses that seem fresh, compared with scavenged and decomposed carcasses. The abundance and types of scavengers and predator populations can be used to predict how long carcass remains might be expected to persist. Ambient temperatures will determine speed of decomposition. These observations should be included in the history.

Species Affected

Much can be learned by knowing which species are dying. Those species present but unaffected are especially important to note because some diseases have a narrow host range and others infect a wide variety of species. For example, duck plague affects only ducks, geese, and swans, whereas avian cholera attacks a more diverse group of bird species. Certain mammalian diseases that kill carnivores may have no effect on other groups.

Age

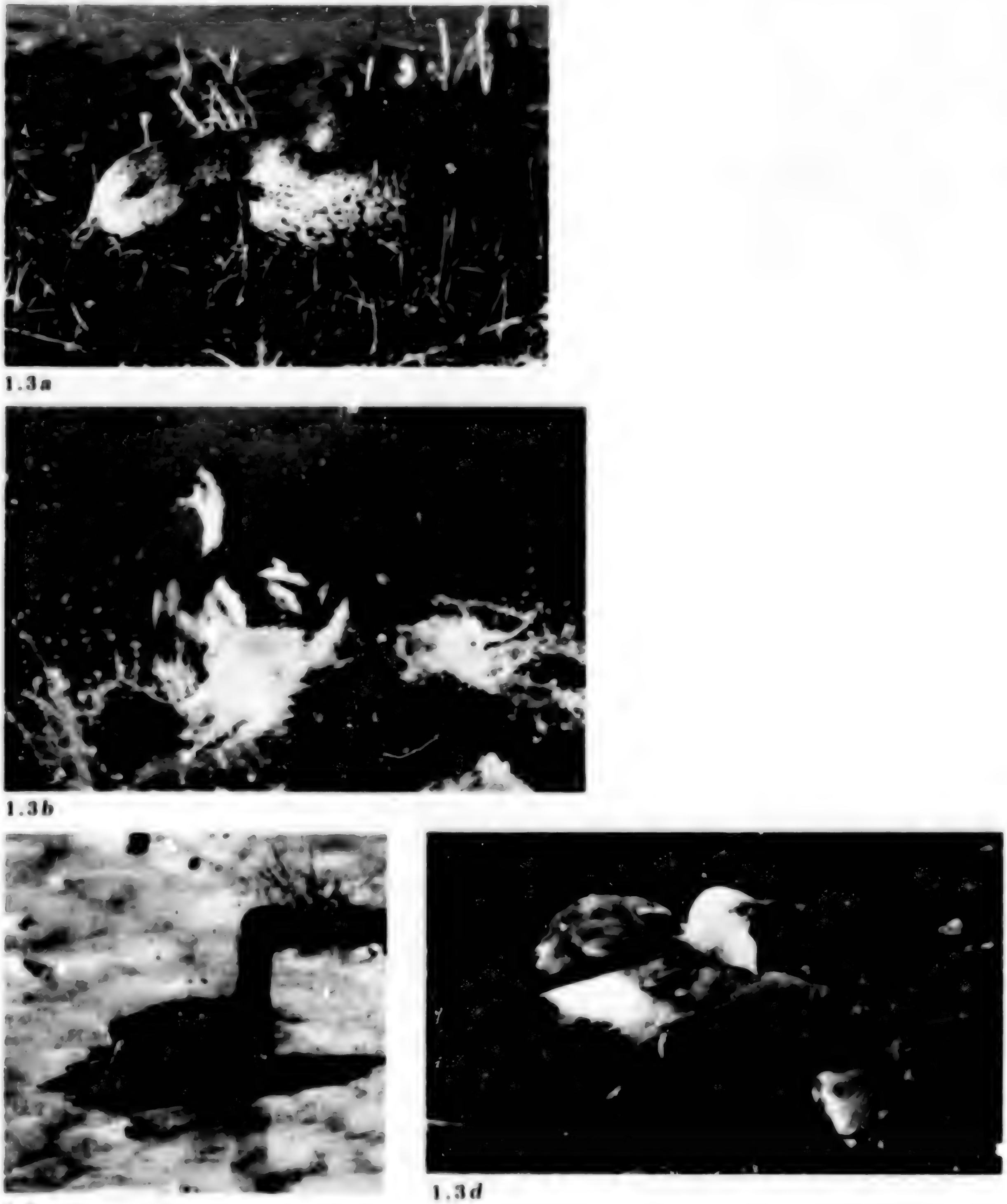
Some disease agents may kill young animals but leave adults unaffected because of age-related resistance; others kill animals of all ages, although young or old animals may be more susceptible because of additional stress placed on these age-groups. When toxins are involved, differences in food habits may result in exposure of young, but not of adults, or vice versa.

Sex

Sex differences in mortality may be apparent in colonial nesters when females are incubating eggs or in other situations where sexes are segregated.

Number Sick Or Dead

The longer a disease takes to kill, the more likely it is that substantial numbers of sick animals will be found. For example, more sick birds will probably be observed during an avian botulism die-off than during an outbreak of a more acute disease such as avian cholera.



1.3*a*

1.3*b*

1.3*c*

1.3*d*

Fig. 1.3. Use of photography to illustrate examples of field observations associated with wildlife morbidity and mortality. (*a*) Undisturbed birds or those approached quietly often remain motionless along the water's edge with the head hanging down. (*b*) When startled, these birds attempt to escape by propelling themselves across the water or land by using their wings because the power of flight has been lost as has the use of their legs (*c* and *d*). *M.F.*

Clinical Signs

If you observe sick animals, describe the clinical signs in as much detail as possible. Describe any unusual physical features such as the animal's response to being approached. Photographs of various behaviors or conditions associated with a disease can be especially useful (Fig. 1.3).

Population at Risk

Try to determine how many animals and what species are in the vicinity of the die-off.

Population Movement

Recent changes in the number of animals in the area should be noted and recorded as well as species present. If animal numbers have increased, try to determine where the animals came from; if numbers have decreased, try to find out where they went. This can often be accomplished by telephone when population movements are being monitored for census, hunting forecasts, and other purposes. Contacts with State, Federal, and private refuge personnel and other resource managers are good primary sources of information.

Specific Features of Problem Area

Location should be described in a manner that allows a relatively specific area to be identified on a road map. Identify where sick and dead animals are found. Those found in agricultural fields may be victims of pesticide exposure; lead-poisoned birds usually seek dense cover; animals dying from acute diseases will be found in a variety of situations. Check any relation between specific animal use of the area and the location of affected animals, e.g., roost sites, loafing area, and feeding sites.

If follow-up investigations are conducted after specimens have been submitted, the findings and observations of those investigations should be summarized as a supplemental report to the original history. A copy of the new report should be maintained in station files and a copy sent to the diagnostic laboratory where the specimens were sent. Both reports should reflect the dates of the investigations, methods used (i.e., low-level fixed-wing aircraft flight and ground searches with retrieving dogs), intensity of efforts (i.e., six people and two dogs performed ground searches for 3 hours), and conditions (i.e., searches during late afternoon, weather overcast, and temperature about 30°F).

The insight provided by good specimen history data and field observations is invaluable to disease specialists. This information enhances understanding of the ecology of disease, thereby serving as a basis for developing ways to prevent future die-offs or to reduce the magnitude of losses that might otherwise occur.

Chapter 2.
Specimen Collection and Preservation

J. Christian Franson

Specimens, as discussed in this handbook, have but a single purpose—to provide information leading to the diagnosis of a cause of disease or death. A specimen may be an intact carcass, various tissues removed from carcasses, or parasites. In any event, the specimen should be as fresh and undamaged as possible.

Choosing a Specimen

When size of the carcass and its condition permit, the best specimen for submission is an entire carcass. This allows the diagnostician to make an assessment of all organ systems and to use appropriate organs for diagnostic workup. Obtain the best specimens possible for necropsy; decomposed or scavenged carcasses are usually of limited diagnostic value. Ideally, one should collect a combination of sick animals, euthanized after clinical signs have been observed, plus a number of the freshest carcasses available. The method of euthanasia should not compromise the diagnostic value of the specimen (see chapter on Euthanasia Methods). More than one disease may be affecting the population simultaneously and, if both sick and dead animals are collected, chances will be increased for detecting multiple diseases. Specimens submitted should be representative of the species involved. If a variety of species are affected, collect several of each; try to obtain a minimum of five specimens per species.

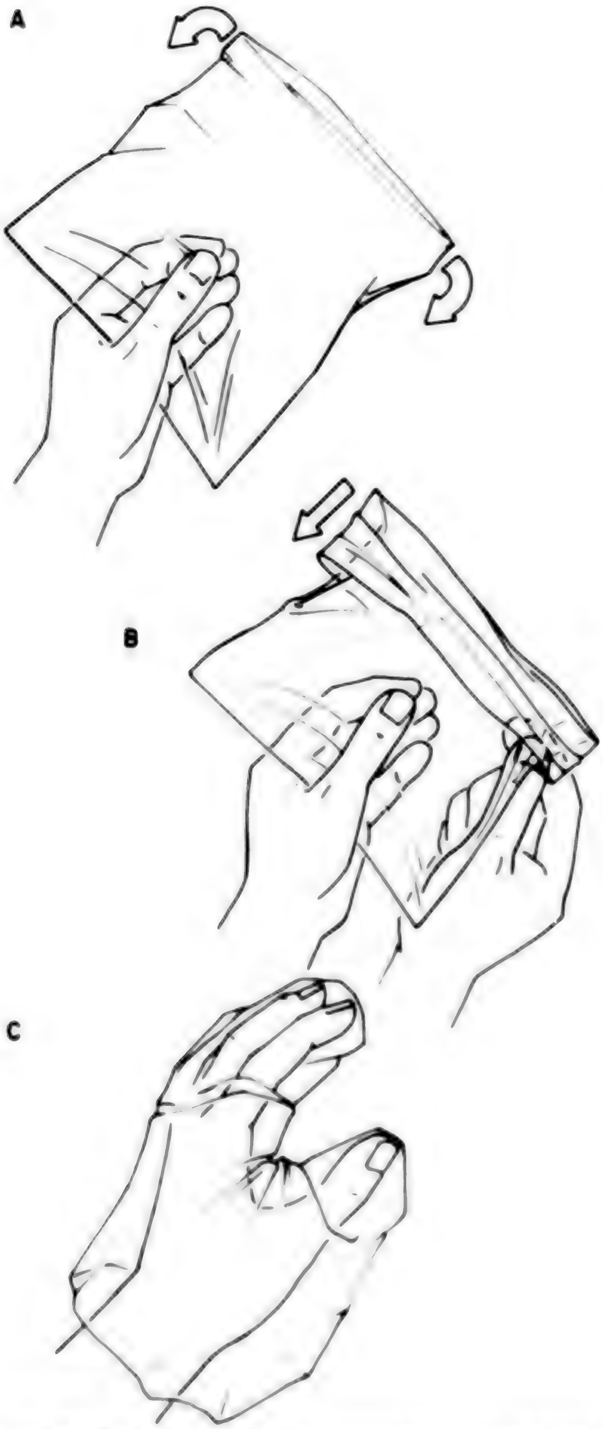
Dissection

If it is impossible to submit an entire carcass, as with large birds or mammals, appropriate organs must be removed and submitted. *If possible, carcasses should not be dissected in the field without first consulting disease specialists* regarding methods of dissection and preservation of tissues or parasites. Help is available from a variety of sources (Appendix B). Field personnel should become familiar with these sources and what specific types of assistance they can provide before an emergency arises.

When collecting carcasses or tissues for diagnosis, the primary consideration should be personal safety. Many mammalian and several avian diseases are transmissible to humans and every carcass should be treated as a potential health hazard. Wear disposable rubber or plastic gloves, coveralls, and rubber boots. If gloves are not available, inverted plastic bags may be substituted (Fig. 2.1). Before leaving an area where carcasses are being collected, double-bag gloves and coveralls and disinfect boots and the outside of plastic bags with a commercial disinfectant or chlorine bleach. Specimens should also be double-bagged in plastic before removal from the area. These precautions will help protect you and minimize transmission of disease to unaffected wildlife populations.

We recommend the use of Whirl-Pak bags for collecting specimens. These bags have a sterile interior, are easy to carry in the field, and can be used for a variety of samples (Fig. 2.2). Identify the specimen by writing directly on the bag with a fine-point indelible marker such as a "Sharpie".

If lesions are noted, it is advisable to collect tissue samples for microscopic examination in addition to samples for microbiology, toxicology, and other assays. With a sharp knife or scalpel, cut a thin (1/8 inch; 3—4 mm) section of tissue that includes both lesion material and adjacent normal-appearing tissue (Fig. 2.3). Take care not to crush tissue in or around the lesion. The tissue sample section should be placed in a volume of 10% buf-



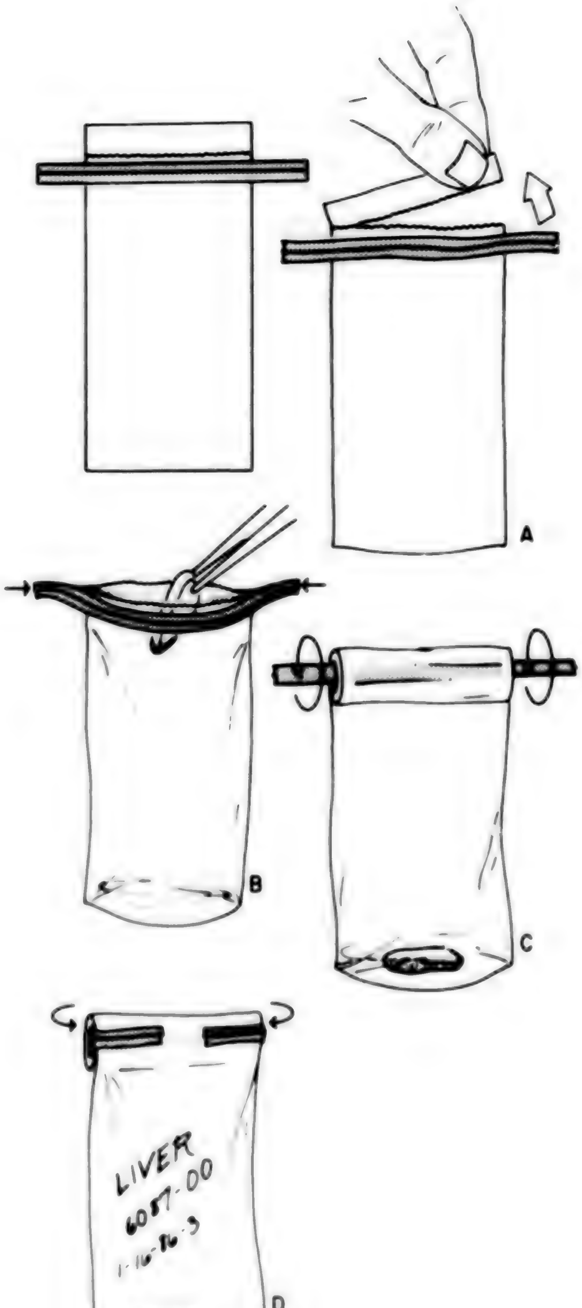


Fig. 2.1. Use of a plastic bag to protect hands from direct contact with animal tissues during specimen-taking if plastic or other waterproof gloves are not available. (*a*) Grasp bag at the bottom and (*b*) with other hand (*c*) pull open end down over hand holding bag. Repeat for the "unbagged" hand. Reversing this process when handling small specimens will automatically place specimens in bag, which then need only be sealed and put into a second bag for packaging and shipment.

Fig. 2.2. Using Whirl-Pak bag for specimen collection. (*a*) Remove top at perforation. (*b*) Open bag by simultaneously pushing the protruding wire-reinforced tabs toward the center to insert the specimen and any appropriate preservative. (*c*) Close bag by pulling on tabs and then twirling bag while holding tabs. (*d*) Secure the closure by folding tabs around bags and label bag with type of specimen, date, and any identifying numbers.

fered formalin solution equal to at least 10 times the tissue volume for adequate fixation. Jars (pint or quart canning jars work well) are convenient containers for fixation, but breakage is a problem in the field. Wide-mouth plastic bottles, such as those made by Nalgene, eliminate the breakage problem and should be part of any field kit (Fig. 2.4). After 2 or 3 days of fixation, tissues can be transferred to Whirl-Pak bags containing enough formalin to keep them wet. Identify the specimen with indelible marker, writing directly on the bag, and pack the bags for shipment so as to prevent tissues from being crushed.

If it is necessary to collect a blood sample from a live bird (for example, if botulism is suspected) or a small mammal, and syringes and needles are not available, sever the head from the neck with a hatchet or large knife and collect the blood in a large-mouth plastic jar. Plastic bottles, Whirl-Pak bags, and 10% buffered formalin can be purchased from laboratory supply houses such as those listed in Appendix C.

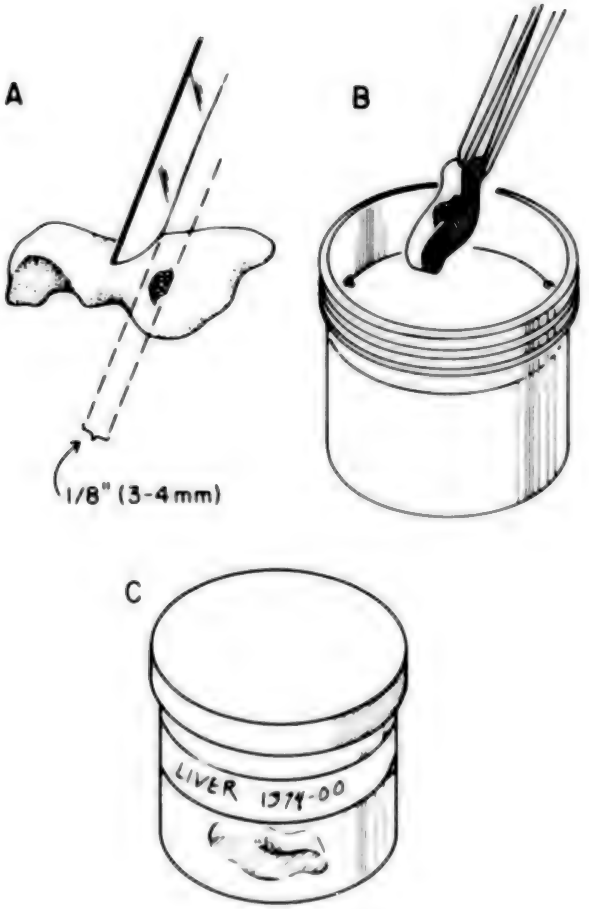


Fig. 2.3. Tissue sample collection for histological examination. (*a*) Tissue sample should include lesion, such as spots in liver, plus some healthy-appearing tissue. The sample must be no thicker than 1/8 inch to ensure adequate chemical fixation by preservative. Use as sharp an instrument as possible (scalpel, knife, razor) for a clean cut. (*b*) Place tissue sample into container of 10% buffered formalin or other suitable fixative or preservative. The volume of formalin in the container should be about 10 times the amount of tissue sample. (*c*) Complete the process by securing the lid and properly labeling the container.

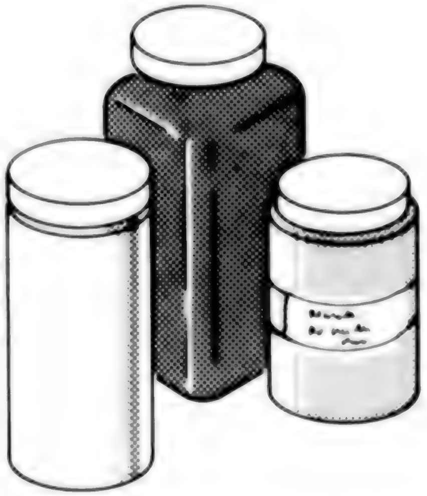


Fig. 2.4. Various plastic bottles used for tissue specimens. Regardless of size or shape, specimen bottles should have a wide mouth and threaded caps for secure closure.

Birds

To dissect a bird, make a midline incision over the keel (Fig. 2.5*a*), peel the skin back, cut through the ribs, and lift the breast muscle and sternum to expose the viscera (Fig. 2.5*b*). You will be able to see the esophagus, heart, liver, and gizzard (Fig. 2.5*c*). Remove any abnormal-appearing tissues or organs, and submit for examination. In general, heart, liver, gastrointestinal tract, and brain should be collected. Keeping tissues as clean as possible, grasp one of the large vessels at the base of the heart, dissect it free (Fig. 2.5*d*) and place in a Whirl-Pak or a clean plastic bag. Remove the liver (Fig. 2.5*e*) and place in another bag. Then grasp the esophagus, tie it off at top and bottom to avoid spilling the contents, dissect it free, and remove the entire gastrointestinal tract including cloaca (Fig. 2.5*f*). Remove the entire head and place it in a fourth bag. All these individually bagged specimens from a single carcass should then be enclosed in one larger bag labeled with the specimen number.

If pesticides, mycotoxins, or other contaminants are suspected as causes of mortality, it may be important to collect environmental samples. Grain (about a handful), or water (1–2 cups) can be placed in plastic bottles or Whirl-Pak bags and handled the same as tissues.

Mammals

Dissection procedures for small mammals are essentially similar to those for birds. However, differences in anatomy will result in different appearance of some internal organs; for example, the presence of a stomach rather than a gizzard, the presence of a diaphragm rather than air sacs, and differences in the shape and size of such organs as the spleen, gallbladder, and kidneys.

Dissection of large mammals is more complex and is beyond the scope of this chapter. Readers are referred to the chapter on Postmortem Examinations in *Wildlife Management Techniques* (Cowan and Karstad 1969) as a general guide for large mammal dissections. The gross internal anatomy of a white-tailed deer is provided as a reference for comparative purposes in Appendix D.

Labeling Specimens

Carcass

Identify each carcass separately with a manila tag fastened by wire to a leg (Fig. 2.6). If such tags are not available, use a 3 × 5 inch card placed inside the bag with the carcass. Information on the tag should include name, address, and telephone number of submitter, collection site, species, whether the animal was found dead or was euthanized (indicate method), and a brief summary of any clinical signs. Use soft pencil or indelible marker, never ballpoint pen. Place each tagged carcass in a separate plastic bag and seal.

Tissues and Organs

For labeling specimens stored in plastic bottles, jars, and tubes, wrap a piece of adhesive tape (masking tape can be substituted) entirely around the container, and use an indelible marker to write on the tape; be sure to list the type of animal from which the sample was taken, the kind of tissue, and the date the sample was taken. Plastic bags should be labeled with the same information but labeling should be directly on the bag.

Specimen Preservation

Either chill or freeze all specimens (use refrigeration, blue ice packs, block ice, or ice cubes), depending on how long shipping to a diagnostic laboratory will take. Freezing reduces the diagnostic usefulness of carcasses, but if they must be held 2 days or more, freezing as soon as possible after collection reduces decomposition. Formalin-fixed tissues should not be frozen. (See chapter on specimen shipment for detailed instructions on packing specimens and shipping them.)

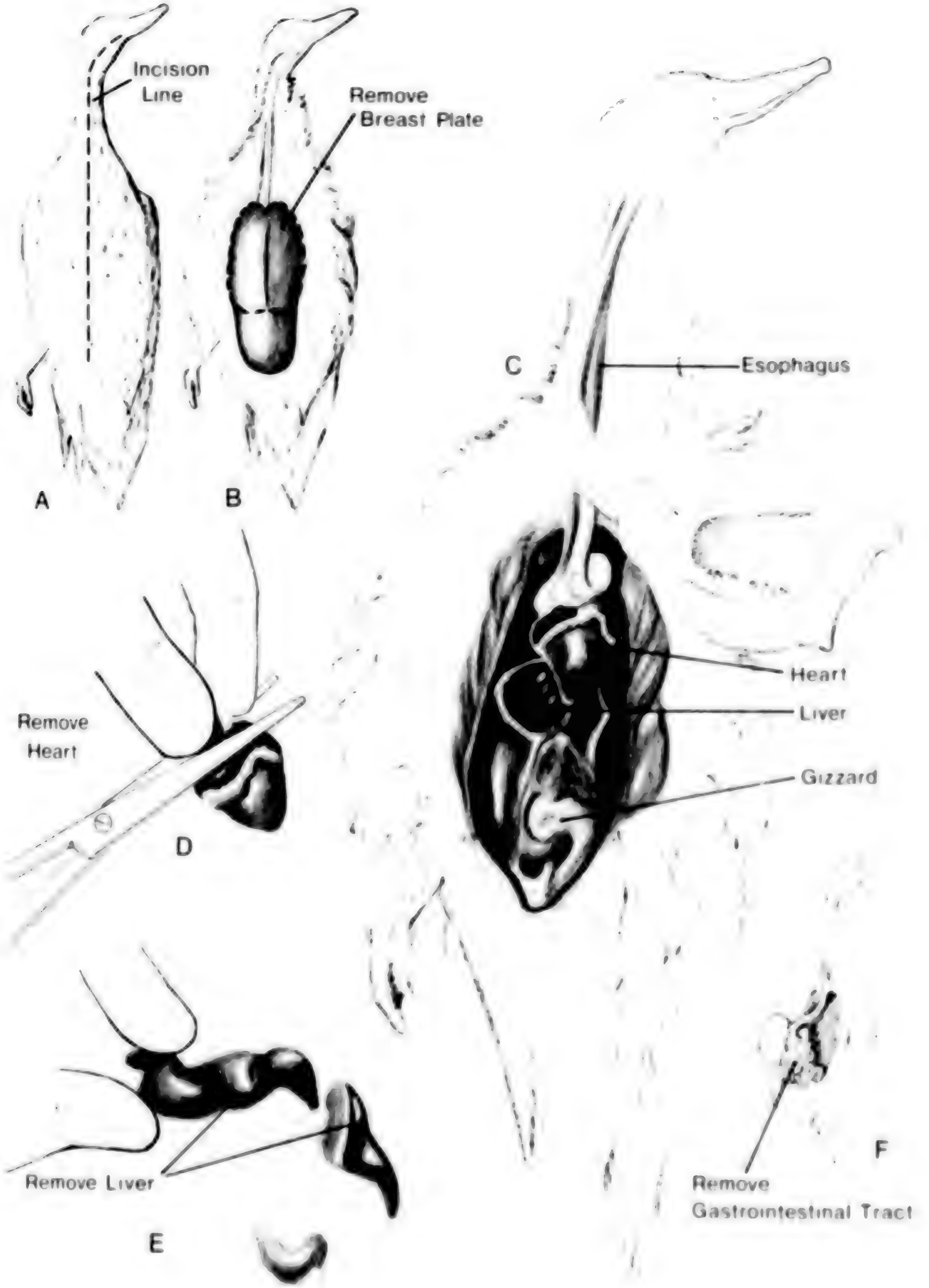


Fig. 2.5. Dissecting a duck carcass (*a*) incision line; (*b*) expose organs under breast muscle and sternum; (*c*) esophagus heart, liver, and gizzard; (*d*) dissect out heart; (*e*) remove liver; and (*f*) tie off and remove the gastrointestinal tract

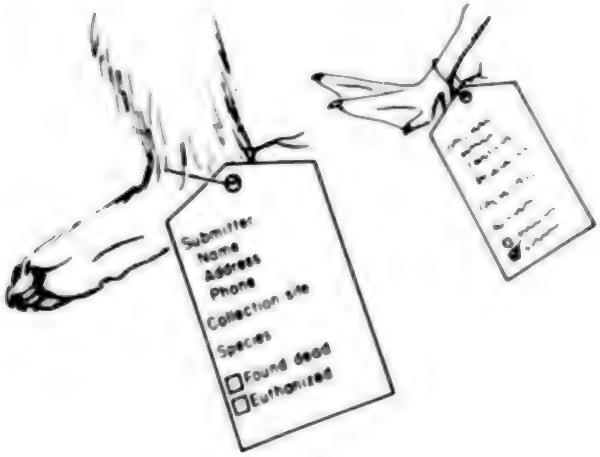


Fig. 2.6. Proper tagging of specimen. History of the specimen (see text for details) should be placed on back of tag.

Supplementary Reading

Cowan, I. M. and L. Karstad. 1969. Postmortem examinations. Pages 251–258 *in* R. H. Giles, Jr., editor. Wildlife management techniques. 3rd edition. The Wildlife Society, Washington, D.C.

Chapter 3.
Specimen Shipment

J. Christian Franson

Procedures for shipping specimens vary with different disease diagnostic laboratories. Therefore, it is important to contact the receiving laboratory and obtain specific instructions. This will facilitate processing of specimens when they reach the laboratory and assure that the quality of specimens is not compromised. Time spent on field investigation, specimen collection, and obtaining an adequate history will be of little value if specimens become contaminated, decomposed, or otherwise spoiled enroute to the diagnostic laboratory.

There are five bases of proper specimen shipment: (1) prevent cross-contamination from specimen to specimen, (2) prevent decomposition of the specimen, (3) prevent leakage of fluids, (4) preserve individual specimen identity, and (5) properly label the package. Basic supplies needed for specimen shipment are shown in Fig. 3.1.

Preventing Breakage and Leakage

Isolate individual specimens from one another by enclosing each one in separate packaging, such as a plastic bag, within the shipment container. Specimens should also be protected from direct contact with any coolant used (e.g., wet ice or dry ice), and all materials within the package must be contained in a manner that prevents leakage to the outside of the shipment container in the event of breakage (e.g., blood tubes) or thawing (wet ice and frozen carcasses) due to delays in transit.

Containing Specimens

Plastic bags should be strong enough to resist being punctured by materials contained within them and from abrasions resulting from contact with other containers within the package.

Styrofoam coolers, shipped in cardboard boxes, are desirable for their insulating and shock-absorbing qualities. Styrofoam at least 1 inch thick is preferred over thinner material. When possible, select styrofoam coolers that have straight sides. Those that are wider at the top than bottom are more likely to break during transit. This can be avoided by filling the space between the outside of the styrofoam cooler and the cardboard box with newspaper or other packing material (Fig. 3.2). If coolers are not available, sheets of styrofoam insulation can be cut to fit the inside of cardboard boxes.

The purpose of the cardboard box is to protect the styrofoam cooler from being crushed during transit and to serve as containment for the entire package (Fig. 3.3). The strength of the box should be consistent with the weight of the package. Cardboard boxes are not needed when hard plastic or metal insulated chests are used for specimen shipment, but can be used to protect those containers from damage and provide a useful surface for labels and addresses.

Cooling and Refrigeration

Chemical coolants such as blue ice packs (Fig 3.4) are preferable to wet ice because their packaging prevents leakage should they thaw. Ice cubes or block ice may be used if leakage can be prevented. This can be accomplished most easily by filling plastic jugs (milk, juice, cider, and soda containers) with water and freezing them. Tape the lids of these containers closed to prevent them from being jarred open during transit.

Use dry ice to keep materials frozen, but be aware that dry ice will freeze specimens intended to be shipped chilled. Also, the carbon dioxide given off by dry ice can destroy some disease agents. This is of concern when tissues rather than whole carcasses are being shipped. Use of dry ice in shipment is regulated and should be cleared with the carrier before shipping.

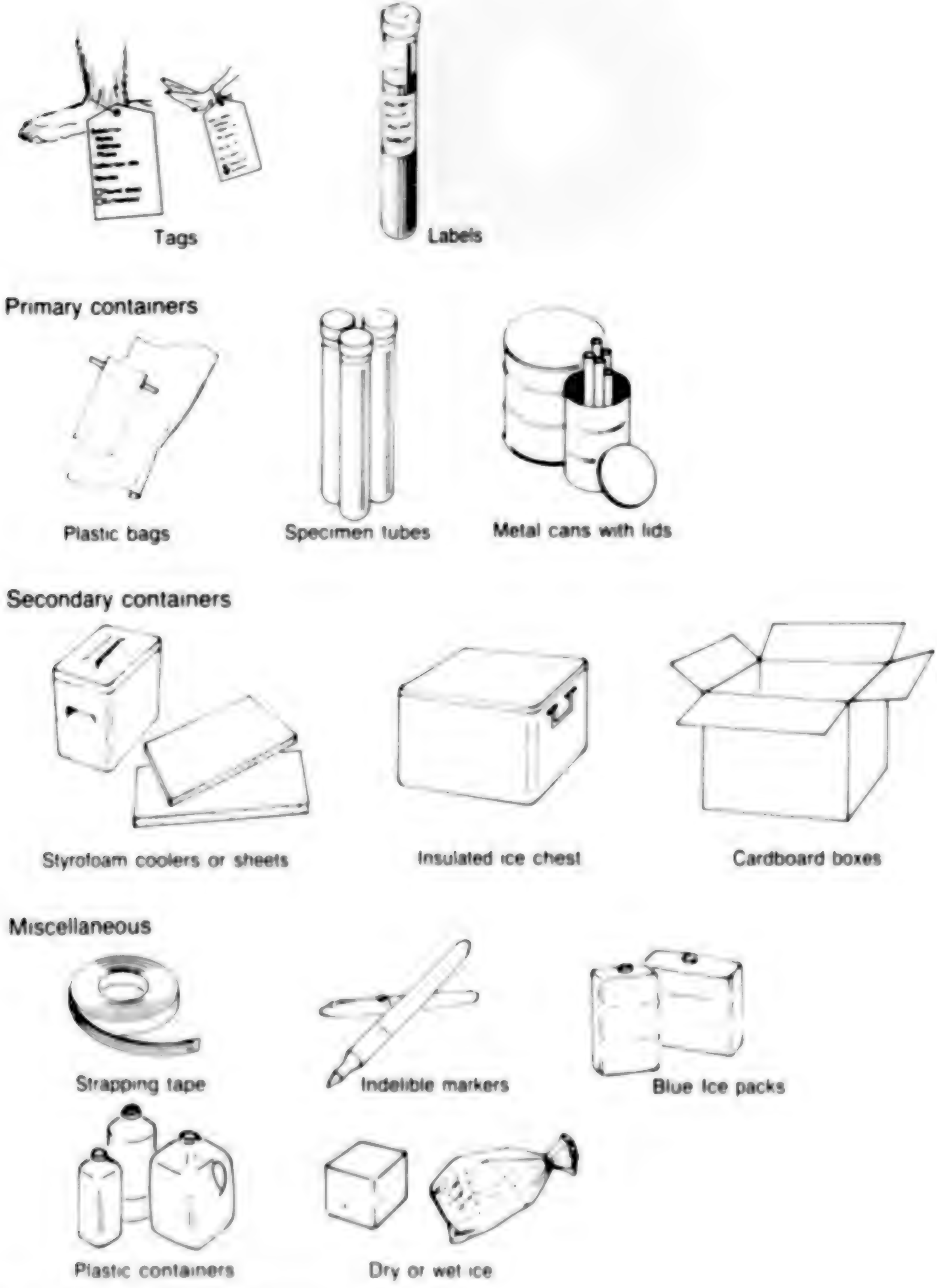


Fig. 3.1. Basic supplies used for specimen shipment

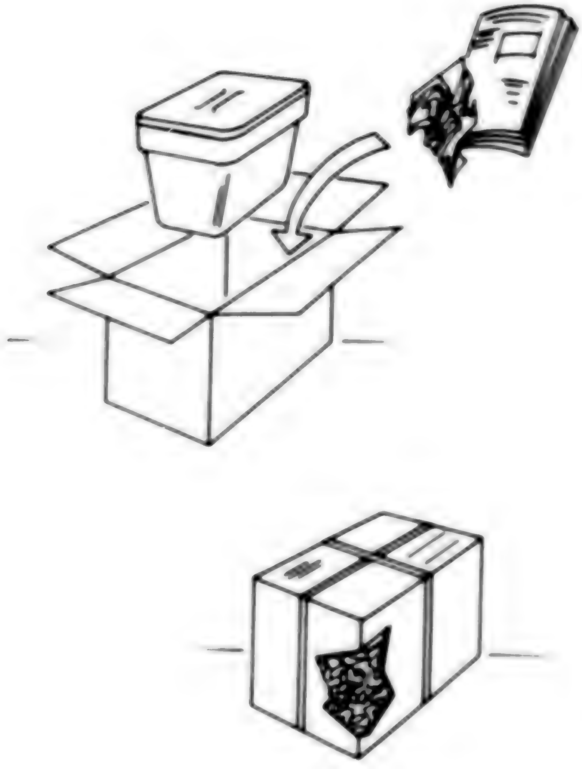


Fig. 3.2. Proper packing to prevent styrofoam coolers from becoming crushed during transit. Place the sealed styrofoam cooler in a sturdy cardboard box. Use crumpled newspaper or other soft packing material to fill all space between the cooler and the box.

Fig. 3.3. Example of the result of improper packaging. *J.R.*

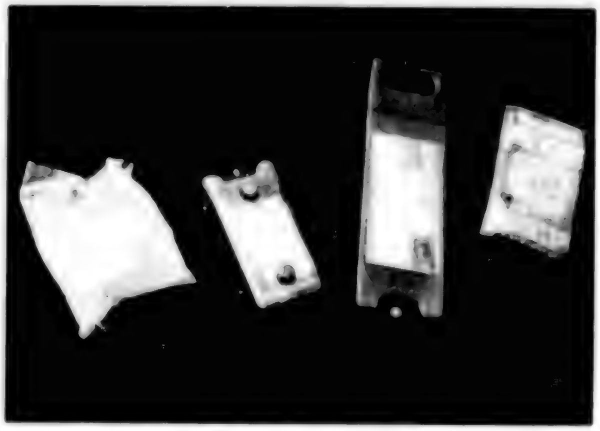


Fig. 3.4. Chemical coolants (blue ice packs) are available in (*a*) soft plastic, (*b*) hard plastic, and (*c*) metal containers. *J.R.*

Preparing Specimens for Shipment to the National Wildlife Health Center (NWHC)

(Other disease diagnostic laboratories may require minor variations in shipping procedures.)

1. Call the NWHC Resource Health Team (608-264-5422, FTS 364-5422) to determine the optimal type and number of specimens for diagnostic procedures, how these specimens are best preserved during transit (e.g., chilled vs. frozen), and when they should be shipped. In most instances, the Resource Health Team asks that specimens be shipped the same day or within 24 hours, but in some instances shipment may be delayed to facilitate specimen receipt and processing.

2. Double-bag carcasses (Fig. 3.5) and place them in a styrofoam cooler lined with a plastic bag. When submitting both frozen and fresh whole carcasses in the same container, you can use the frozen carcasses as a refrigerant to keep the fresh carcasses chilled by interspersing individually bagged frozen carcasses among the individually bagged fresh carcasses or by placing the fresh carcasses between two layers of frozen carcasses (Fig. 3.6).

Blood tubes and other breakable containers of uniform size can be protected by packing them in a common plastic bag that is sealed within a coffee can (Fig. 3.7). Pack any space around the specimen containers within the coffee can (side and top) with paper or some other absorbent material to prevent jarring that could cause breakage, and to collect fluids if breakage does occur. Then, seal the coffee can within a plastic bag before placing it in the styrofoam cooler.

3. When using blue ice packs, intersperse them among specimens; place other types of coolants in locations within the styrofoam container that will provide maximum cooling for all contents or keep everything frozen when dry ice is used (Fig. 3.8). Fill all empty space within the styrofoam cooler with newspaper to prevent materials from being tossed around during transit. The insulating properties of newspaper will also help maintain cool temperatures within the package and its absorbent qualities will help prevent leakage of fluids to the outside of the box or container.

4. Close the plastic bag lining the cooler and seal the lid with strapping tape (Fig. 3.9). Tape the specimen data sheet and history, contained in an envelope within a waterproof plastic bag, to the top of the cooler (Fig. 3.10*a*).

5. Enclose the styrofoam cooler in a cardboard box and secure the contents with strapping tape (Fig. 3.10*b*).

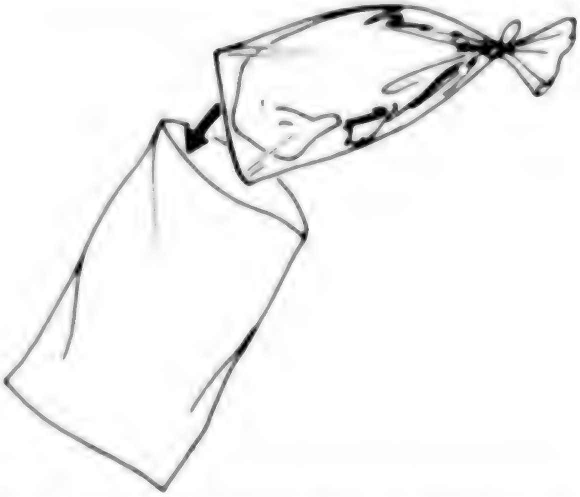


Fig. 3.5. Individual carcasses should be double-bagged to prevent leakage of fluids and cross-contamination of specimens.

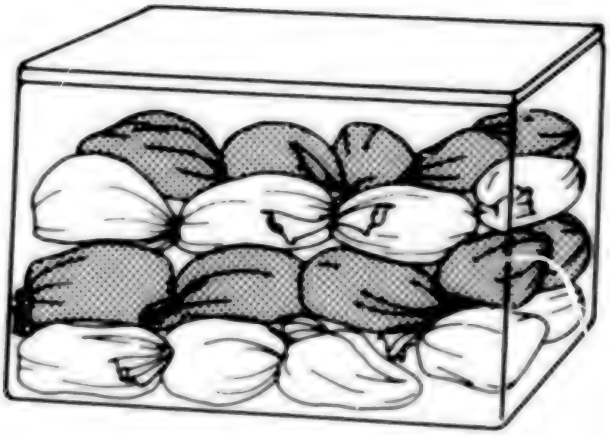


Fig. 3.6. Frozen carcasses (white bags) can be used to keep fresh specimens (dark bags) chilled during short transit times (24 hours or less). Fill the space between the carcasses and the top of the container with newspaper to provide additional insulation to maintain the cold.

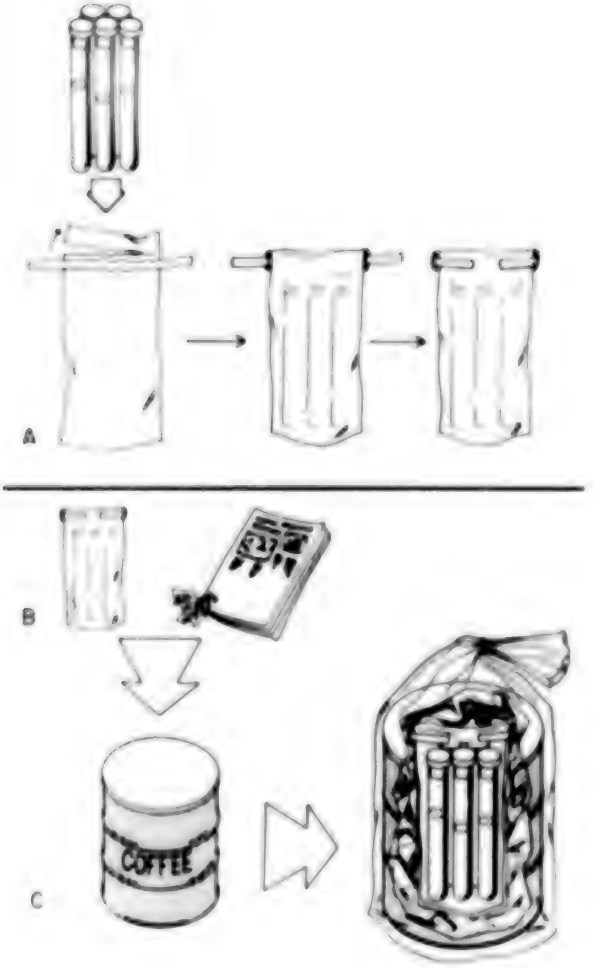


Fig. 3.7. Packing sequence of (*a*) blood tubes being packaged within Whirl-Pak or other plastic bag; (*b*) bag then being placed in metal can or hard plastic container and packed with crumpled newspaper or other absorbent, soft, space-filling material; and (*c*) the can being enclosed in a plastic bag which is then sealed.

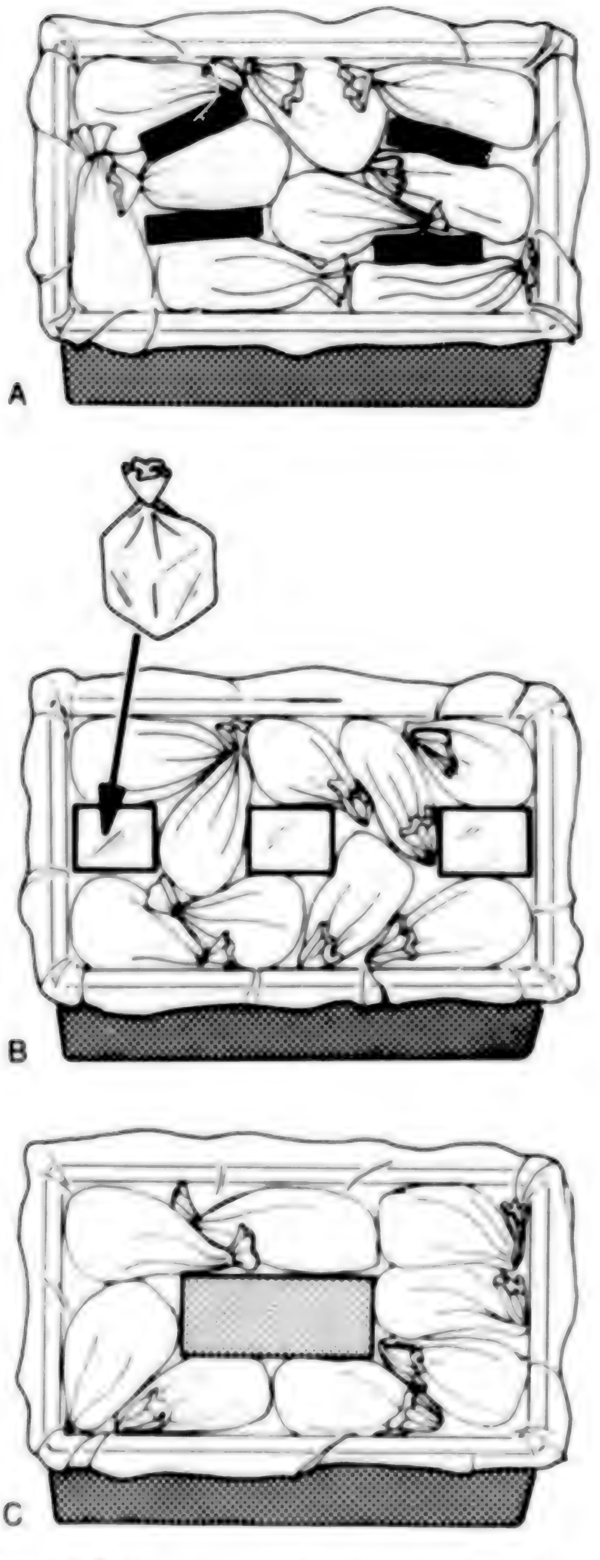


Fig. 3.8. Packing specimens for shipment when *(a)* blue ice packs, *(b)* wet ice, and *(c)* dry ice are used as coolants. Note that in each instance, the shipping container is lined with a large plastic bag.

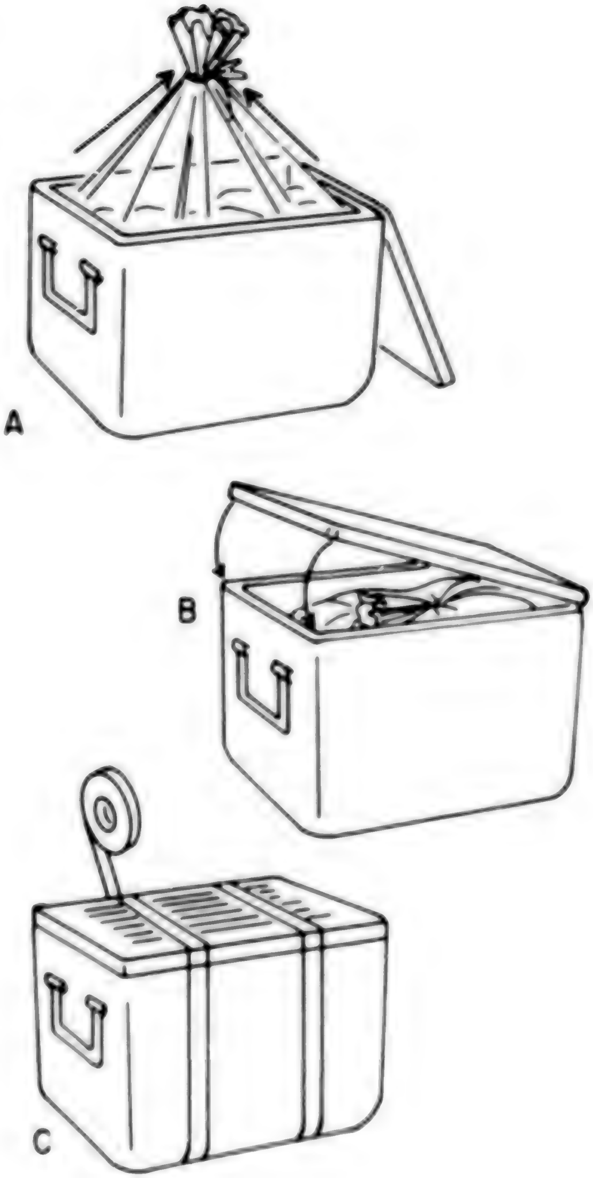


Fig. 3.9. Closure of specimen container. *(a)* Secure the large plastic bag containing the specimens by tying the tip; *(b)* close the cooler lid and *(c)* secure the container with several bands of strapping tape.

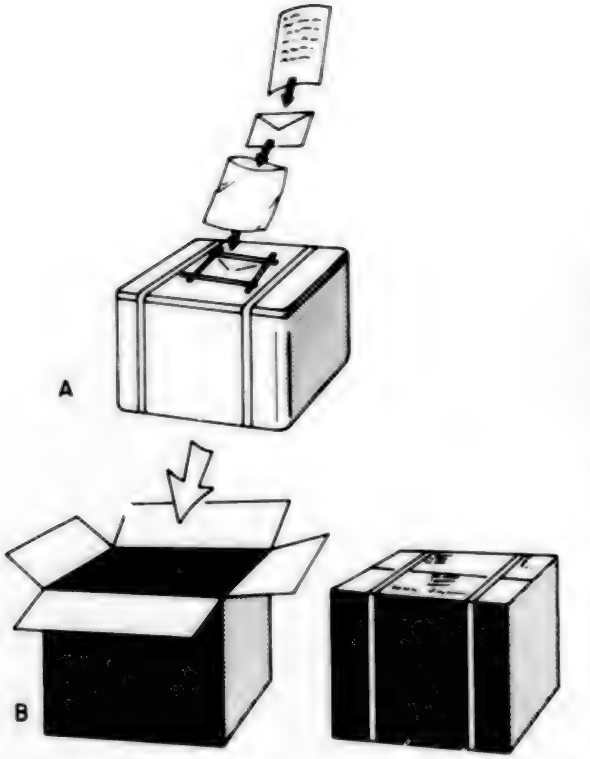


Fig. 3.10. Completion of the packing process. *(a)* Tape specimen data sheet and history, contained in an envelope within a waterproof plastic bag, to top of cooler. *(b)* Place cooler in cardboard box and secure box with several bands of strapping tape.

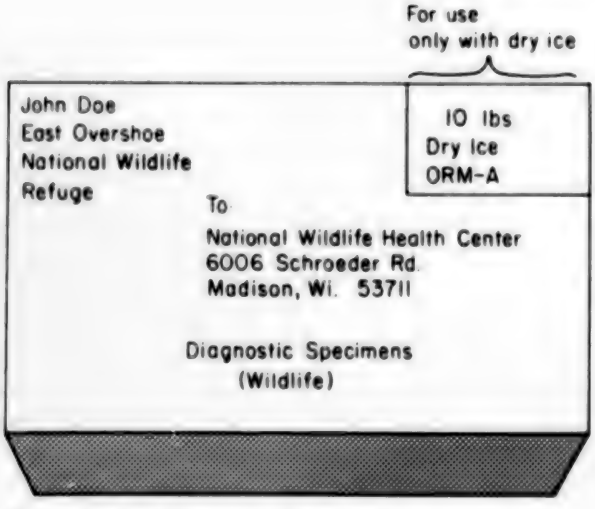


Fig. 3.11. Proper labeling of package.

Federal Shipping Regulations for Packaging and Labeling

Your packaging and labeling of specimens must conform to the following regulations.

Under Fish and Wildlife Regulations (50 CFR 14), containers with wildlife specimens must bear the name and address of the shipper and consignee, and an accurate statement of the contents (by species and number of each species) must be conspicuously marked on the outside of the container. An alternative to this requirement is to conspicuously mark the outside of each package or container with the word "wildlife" or the common names of the species contained within the package. In addition, an invoice or packing list that includes the name and address of the consignee and shipper, and which accurately states the types and quantities of each species contained in the shipment, should be secured to the outside of one container in the shipment.

In addition to Fish and Wildlife Service regulations, the interstate shipment of diagnostic specimens is subject to applicable packaging, labeling, and shipping requirements for etiologic agents (42 CFR Part 72). These regulations do not require identifying diagnostic specimens as etiologic agents when the disease agent is not known or only suspected. However, all specimen packages sent to the NWHC should be prominently labeled with the words "DIAGNOSTIC SPECIMENS." Packaging requirements under 42 CFR Part 72 are met by following the preceding recommendations 2 through 5 for enclosing specimens within two containers before being enclosed within the package.

Hazardous Material Regulations of the Department of Transportation apply whenever dry ice is contained within the shipping container (49 CFR Part 172–173). Clearly write the amount of dry ice contained within the package in the upper right-hand corner on the outside of the container. The letters "ORM-A" must appear under the amount of dry ice and this information must be written within a rectangular area marked on the outside of the package. The words "DIAGNOSTIC SPECIMENS" must appear under that designation. Properly labeled containers for use with and without dry ice are illustrated in Fig. 3.11. Labeling should be done with permanent markers if possible.

Commercial Carriers

Specimens should be shipped by carriers that can guarantee 24-hour delivery to the location of the diagnostic laboratory (for the NWHC, this is Madison, Wisconsin). Air freight is preferred over other types of carriers. Federal Express, Purolator, Emery, DHL, and U.S. Postal Service Express Mail have all proved to be satisfactory in meeting this requirement. For many locations, these commercial delivery services will pick up packages at the point of origin. When you have completed shipping arrangements, recontact the NWHC Resource Health Team to provide the airbill number and estimated time of arrival. This information is needed to allow prompt tracing of shipments that may not arrive on schedule and to schedule work at the laboratory.

Chapter 4.
Disease Control Operations

Milton Friend and J. Christian Franson

Individual disease outbreaks have killed many thousands of animals on numerous occasions. Tens of thousands of migratory birds have died in single die-offs with as many as 1,000 birds succumbing in 1 day. In mammals, individual disease outbreaks have killed hundreds to thousands of animals with, for example, hemorrhagic disease in white-tailed deer, distemper in raccoon, Errington's disease in muskrat, and sylvatic plague in wild rodents.

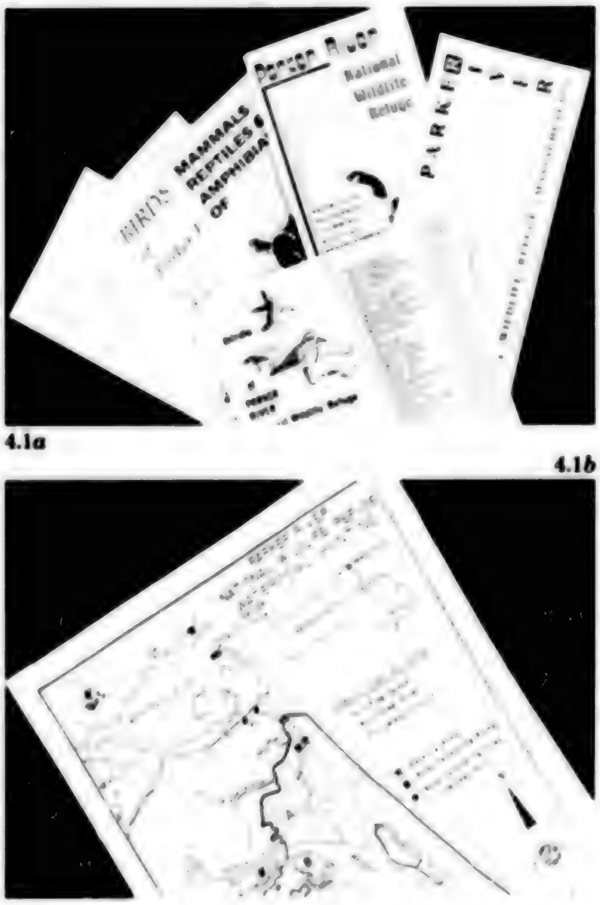
The ability to successfully combat such explosive situations is highly dependent on the readiness of field personnel to deal with them. Because many disease agents can spread through wildlife populations very fast, advance preparation is essential in preventing infected animals from spreading disease to additional species and locations. Carefully thought-out disease contingency plans should be developed as practical working documents for field personnel and updated as necessary. Such well-designed plans can prove invaluable in minimizing wildlife losses and costs associated with disease control activities.

Although requirements for disease control operations vary and must be tailored to each situation, all disease contingency planning involves general concepts and basic biological information. This chapter, intended as a practical guide, identifies the major activities and needs of disease control operations, and relates them to disease contingency planning.

Planning Activities

Identification of Needs

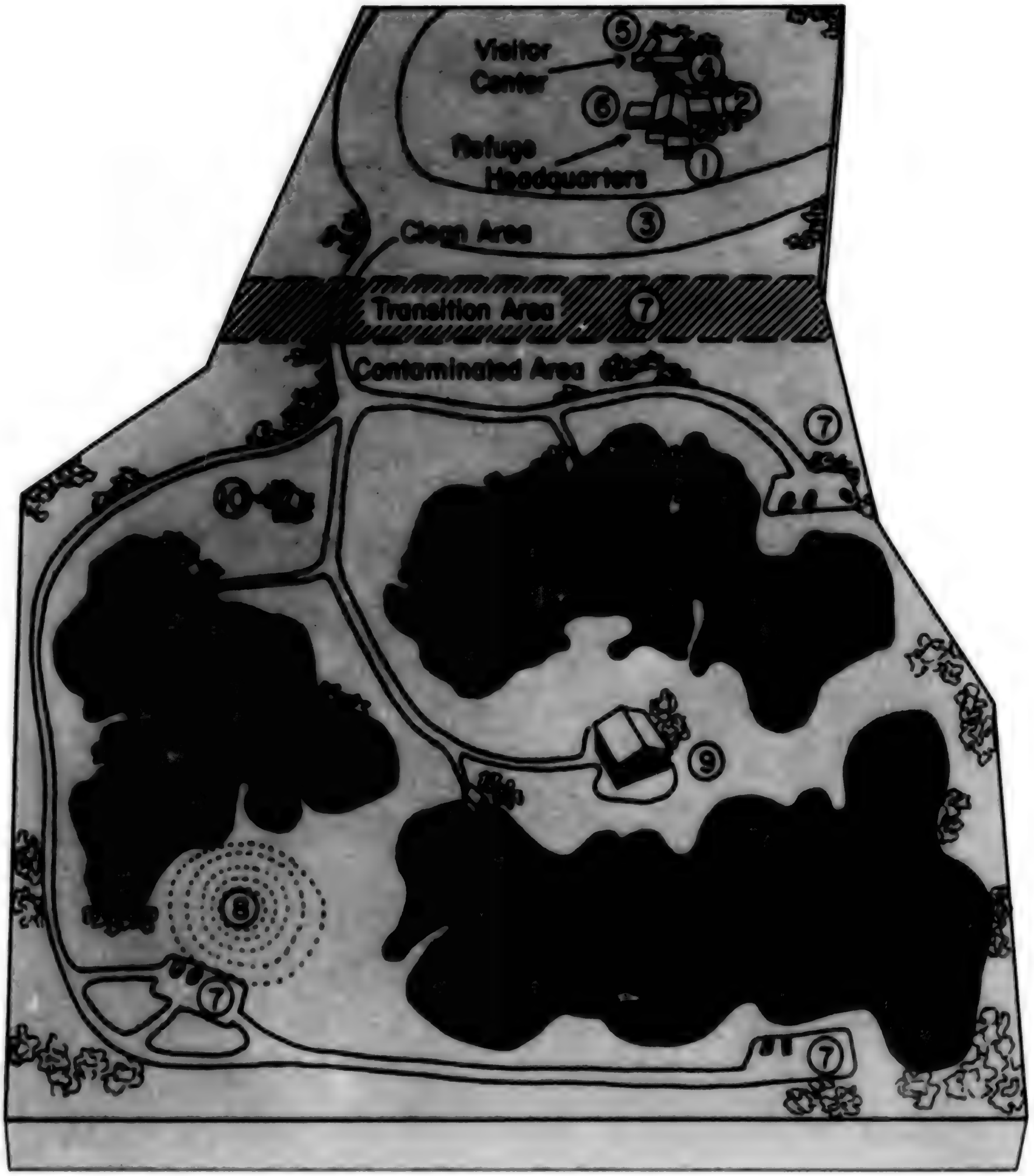
Effective planning for combating wildlife disease outbreaks requires an understanding of disease control operations and the basic needs associated with them (Tables 4.1 and 4.2). This information is the basis of disease contingency planning (Table 4.3; Figs. 4.1 and 4.2).


BIRDS
MAMMALS REPTILES & AMPHIBIANS OF
Parker River National Wildlife Refuge
PARKER RIVER

4.1a

4.1b

Fig. 4.1. (*a*) Station brochures, bird lists, and other public-use documents provide a wide variety of site-specific background information and should be included as part of the station's disease contingency plan. (*b*) Documents containing maps of the area indicating access points provide essential information. *J.R.*



Key

1. Command post—Headquarters administrative area
2. Staff and press briefing room—refuge manager's office
3. Parking
4. Eating area—conference room
5. Staff rest area—visitors' center
6. Equipment and supply receipt—garage
7. Decontamination areas—boathouses, transition areas, parking lots
8. Carcass disposal site—observation hill
9. Animal holding—pole barn (has cement slab and electricity)
10. Laboratory investigations—shed (has cement slab, water, electricity)

Fig. 4.2. Work areas for disease control operations, Bogus National Wildlife Refuge.

Biological Data

All disease outbreaks consist of three main parts: a susceptible host population, a disease agent, and the environment in which the host and agent exist. Disease control involves breaking the connections between these factors. Disease contingency plans expedite these efforts by providing basic information on the distribution and types of animal populations on the area, animal movement patterns, any history of disease problems on the area, and general environmental features. This information along with facts gathered at the time of a disease outbreak provide a profile for biological assessment and a basis for specific disease control actions.

Knowing what types of disease problems have previously occurred on the area, their general locations, the month and year they occurred, species affected, and general magnitude of losses is also of considerable value when responding to a disease outbreak. This information should be displayed in tabular form within the contingency plan (Table 4.4). Animal population data are best represented by simple graphs and charts that convey general characteristics (Fig. 4.3); precise data are not needed. Outline maps are useful for displaying concentration and feeding areas used by wildlife (Fig. 4.4) and major movement patterns (Fig. 4.5). Here again, general information rather than detail is all that is necessary.

Response Activities

Problem Identification

Early detection and rapid and accurate assessment of the causes of disease problems are essential components of effective disease control operations. These are accomplished through surveillance of wildlife populations to detect sick and dead wildlife, and the prompt submission of specimens to qualified disease diagnostic facilities. The speed with which large numbers of wildlife can become exposed to disease agent, and differences in control activities required for different types of disease problems place a premium on both the speed and accuracy of diagnostic assessments. Once a disease problem has been identified, the following basic activities are carried out to various degrees.

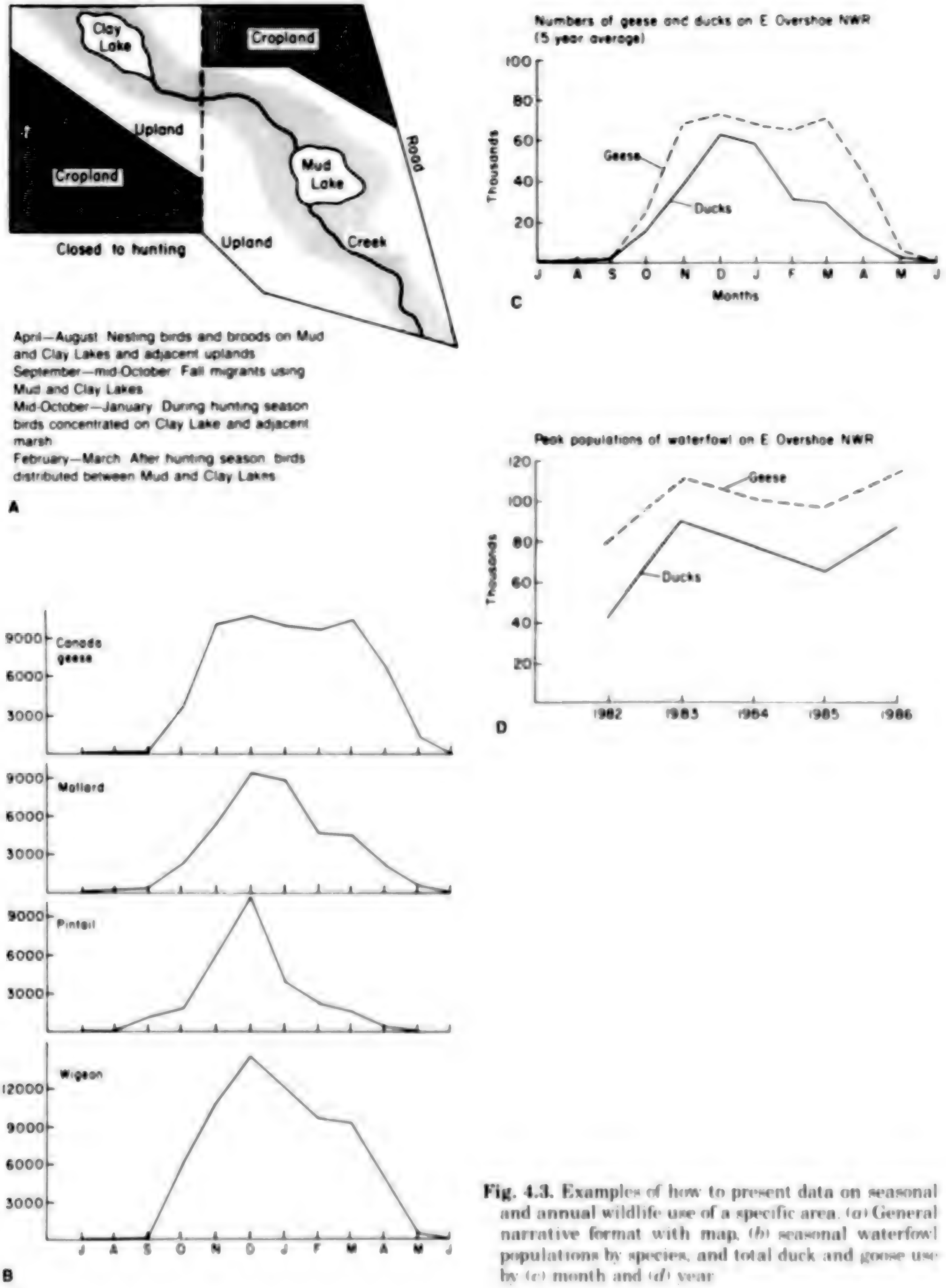
Carcass Removal: Protective Clothing and Supplies

Animals that have died from disease are a primary source of the disease agent and need to be removed from the environment to prevent transmitting the disease to other animals through contact with or consumption of the carcass. Disease organisms released from tissues and body fluids when carcasses decompose also contaminate the environment. Some pathogenic viruses and bacteria can survive for several weeks or longer in pond water, mud, and soil.

Because carcass collection concentrates a lot of diseased material in a small area, it is essential that this material be handled so as not to release infectious agents into the environment or to jeopardize the health of personnel. Great care also needs to be taken to prevent mechanical movement of the disease agent from the problem area to other areas.

Personnel assigned to carcass collection need to wear protective outer garments that can be disinfected and removed before leaving the area. Typically, these include boots, coveralls or raingear, gloves, and a head covering (Fig. 4.6). Raingear are preferred over coveralls, when ambient temperature allows their use, because they can be disinfected before being removed. Instead of stockpiling raingear to be prepared for a major disease outbreak, know where such outerwear can be quickly obtained if needed. In some instances purchase will be necessary, but it may be possible to borrow raingear from the military (including the National Guard) or rent coveralls and other outerwear from commercial sources if disease emergencies occur.

Use disposable coveralls and outer gloves when possible (durability and cost are two major considerations), and remove them before leaving the disease control operations area. Destroy outer garments if they are disposable, or double-bag them before transportation to a location where they can be thoroughly washed before being reused. Here again, focus preplanning on knowing where coveralls and gloves can be quickly obtained rather than stockpiling them for an unpredictable need. Dishwashing gloves, work gloves, and other types of rubber gloves are readily available at hardware and other retail stores, as are a large variety of scrub brushes for cleaning activities (Fig. 4.7).



April—August Nesting birds and broods on Mud and Clay Lakes and adjacent uplands
September—mid-October Fall migrants using Mud and Clay Lakes
Mid-October—January During hunting season birds concentrated on Clay Lake and adjacent marsh
February—March After hunting season birds distributed between Mud and Clay Lakes

Fig. 4.3. Examples of how to present data on seasonal and annual wildlife use of a specific area. (*a*) General narrative format with map, (*b*) seasonal waterfowl populations by species, and total duck and goose use by (*c*) month and (*d*) year

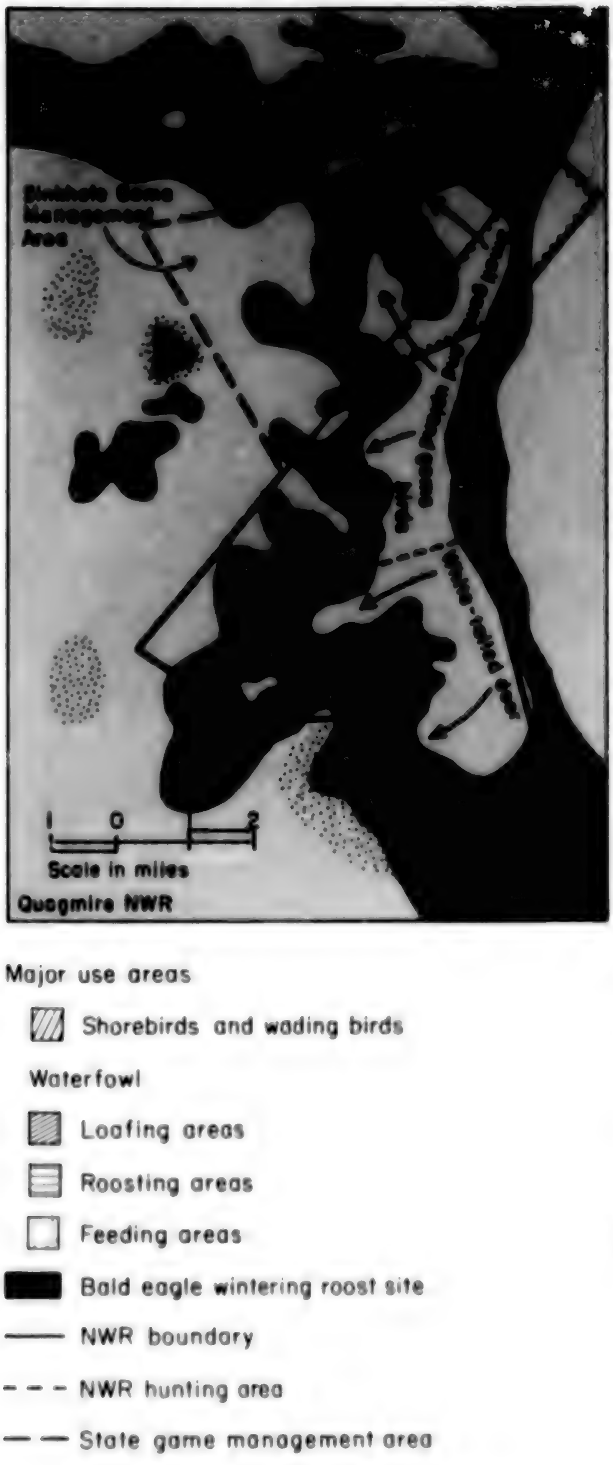


Fig. 4.4. Example of an outline map showing concentration and feeding areas used by wildlife.

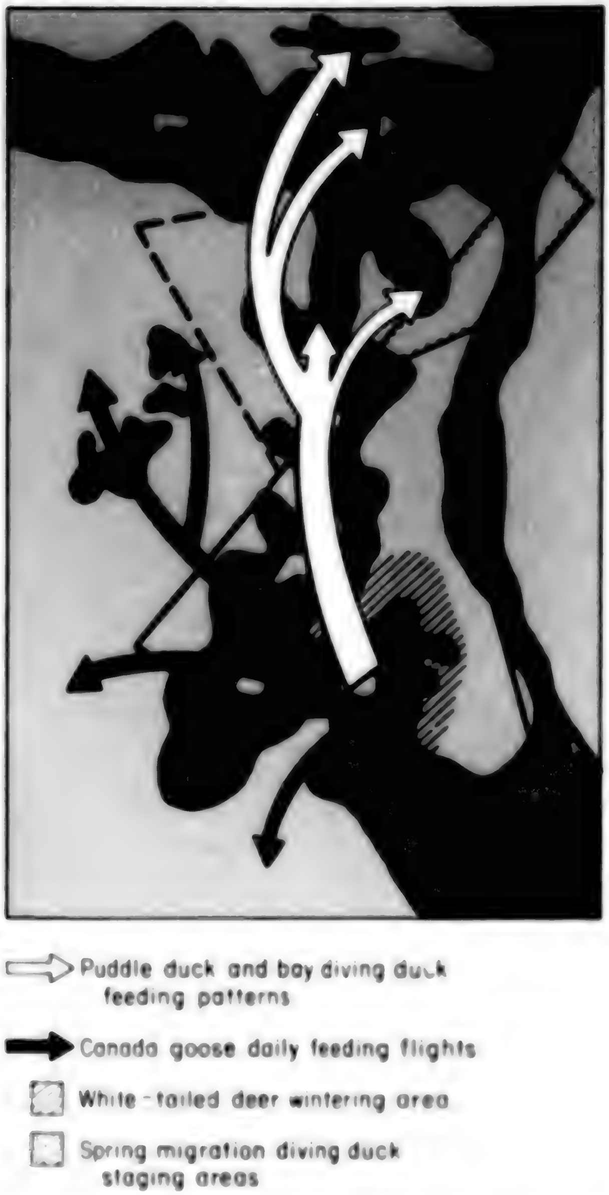
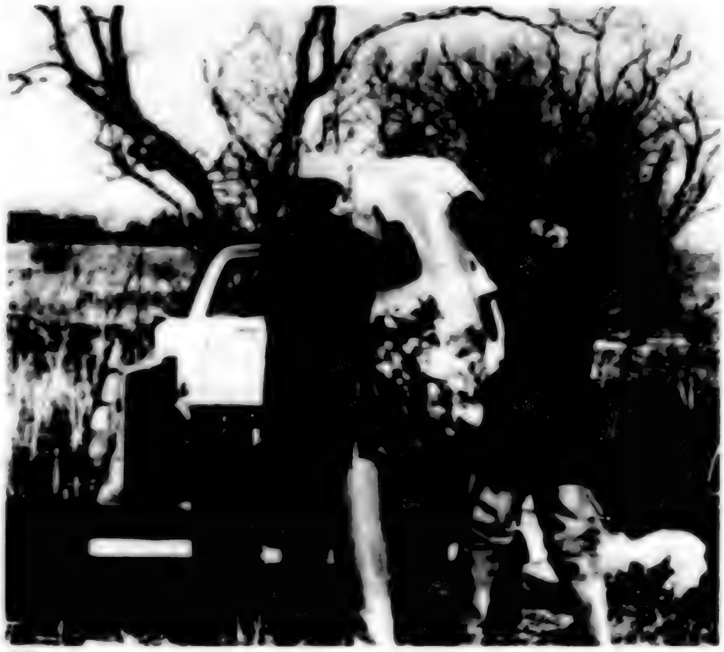

⇨ Puddle duck and bay diving duck feeding patterns
➡ Canada goose daily feeding flights
▨ White-tailed deer wintering area
☐ Spring migration diving duck staging areas

Fig. 4.5. Example of an outline map showing major movement patterns of species.



4.6a

4.6b

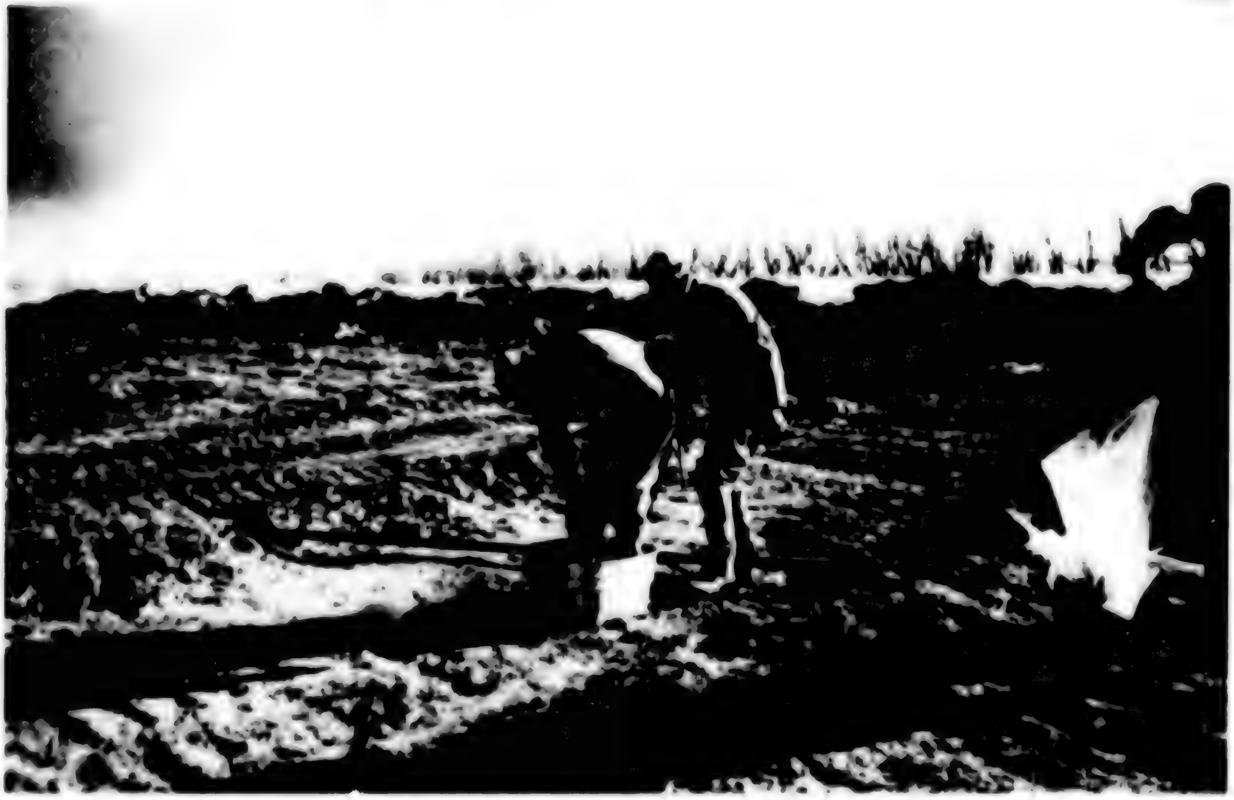


Fig. 4.6. *(a)* Protective clothing such as coveralls, boots, head covering, and gloves should be used for carcass cleanup activities. *(b)* Before leaving the area, boots should be decontaminated and outer clothing removed and bagged for transportation to a location where they can be washed before being reused. *(a)* *W.B.*; *(b)* *M.F.*

Carcass removal activities call for heavy-duty plastic bags or containers. Plastic body bags used by the military are excellent for containment of wildlife carcasses. Plastic garbage cans lined with commercially available heavy-gauge leaf and litter plastic bags are also excellent containers for transporting carcasses. These containers are especially useful when collecting bird carcasses by boat (Fig. 4.8*a*), and for transporting carcasses in truck beds. Bags should be tied shut and the lids secured when transporting these containers to carcass disposal sites (Fig. 4.8*b*).

Depending on conditions, a variety of watercraft (Fig. 4.9) and all-terrain vehicles (Fig. 4.10) are useful for carcass searches and transportation of carcasses to collection and disposal sites. In some instances the expense of helicopters may be warranted. Pickup trucks and other four-wheel vehicles are also indispensable under some field conditions.

Dogs have been used extensively in wildlife management, and are considered a valuable search tool when appropriately chosen and handled. Dogs should be used whenever possible to locate carcasses if there is no disease risk to them. Infectious diseases of wild North American birds do not pose a significant health threat to dogs. However, diseases of carnivores, such as rabies, distemper, and some other mammalian diseases, should be avoided. Disease risk can be determined on a case-by-case basis in consultation with wildlife disease specialists. Local retriever clubs or kennels may be useful sources of dogs.

The contingency plan should identify sources of various equipment, the type of arrangements available (e.g., loan or rental), and the contact persons and their telephone numbers. Commonly used supplies and equipment needed to support disease control operations are summarized in Table 4.2.

Carcass Disposal

The primary goal of carcass disposal is to prevent spread of the disease agent through environmental contamination. Because concentrated amounts of infectious or highly toxic agents are being handled, this activity requires proper training and supervision. Incineration, burying, and rendering are the three basic approaches available.

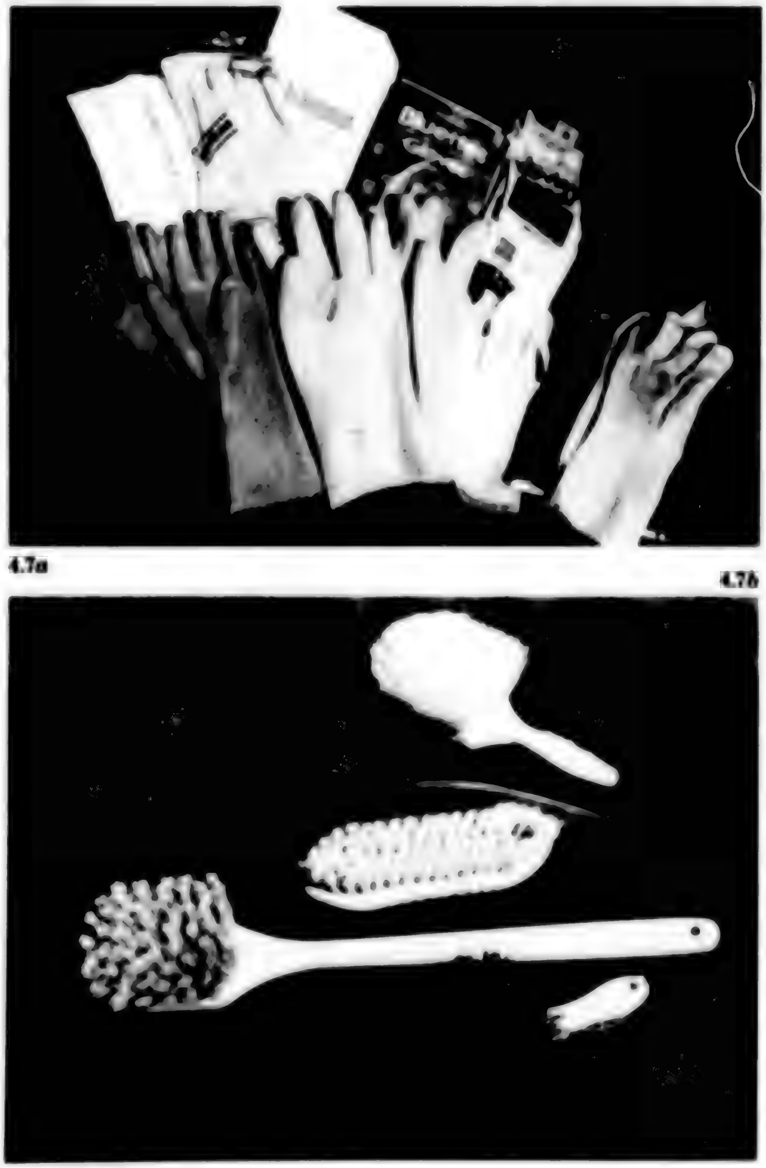
Incineration is generally the preferred method for disposing of carcasses and contaminated materials associated with wildlife disease outbreaks. Consider purchasing or constructing portable incinerators (Fig. 4.11) for areas with recurring disease problems. Portable garbage incinerators can sometimes be borrowed from State parks and other sources. If portable incinerators are not available, open burning with tires, other fuel, or both, may be used depending on local air pollution standards. Carcasses may be burned above or below ground (Fig. 4.12). It is important to keep the fire contained and to get sufficient air movement under the carcasses to maintain a hot fire and get a complete burn. Wood, coal, fuel oil, napalm, and other fuels have been successfully used for this purpose. Never use gasoline because of the hazards involved. You can facilitate incineration by stacking or piling carcasses on the burning platform, soaking them with used oil or some other fuel, and allowing about 10 to 15 minutes for the fuel to soak in before igniting them. The heat generated in large-scale carcass burning operations is intense enough to cause metal pipes to bend (Fig. 4.12*d*). Therefore, a sturdy support surface is needed upon which to place carcasses and avoid collapse of the structure into the fire.

In dry weather conditions, burning in a pit surrounded by a vegetation-free area is more desirable than above-ground burning. In either situation, piling too many carcasses on the fire at one time is a common mistake. Carcasses should generally be burned one layer at a time (Fig. 4.13). When cinder blocks are used to support burning platforms, the length of the platform should be extended to keep the blocks out of direct heat or they will soon crumble. The only limiting factor in disposing of carcasses by burning seems to be the ingenuity of individual field managers and their staffs.

When burning is not feasible or needed, burial is often a suitable alternative. Burial sites must be selected carefully with consideration given to groundwater circulation and drainage, and any potential for later carcass exposure. Sprinkle lime or fuel oil on carcasses to discourage uncovering by scavengers, and cover with at least 3 to 4 feet of soil.

In instances where the combination of animal species, cause of mortality, and local situation allows, carcasses may also be disposed of through an animal rendering plant, and in rare instances infected aggregations of animals may be killed and processed for food. Both of these methods are sometimes used for domestic species and captive-reared wildlife, but conservation laws generally prohibit the processing of free-living wildlife (with the exception of fish) as a commercial food source within the United States. Judgments on the use of rendering and food processing as animal disposal methods should be made only by qualified disease control specialists.

To the extent possible, carcass disposal should take place on-site to reduce the risk associated with transport of contaminated material. Regardless of whether burning or burial is involved, earth-moving equipment is needed. The disease contingency plan should identify how and where bulldozers, backhoes, and similar equipment can be obtained.


4.7a

4.7b

Fig. 4.7. (*a*) Examples of readily available disposable and reusable gloves for disease control operations. Dishwashing gloves, surgical gloves, rubber work gloves, and other types can be purchased at drug and hardware stores and medical and laboratory supply houses. (*b*) A wide variety of scrub brushes needed for decontaminating boots, equipment, and other surfaces are also readily available from local merchants. *J.R.*

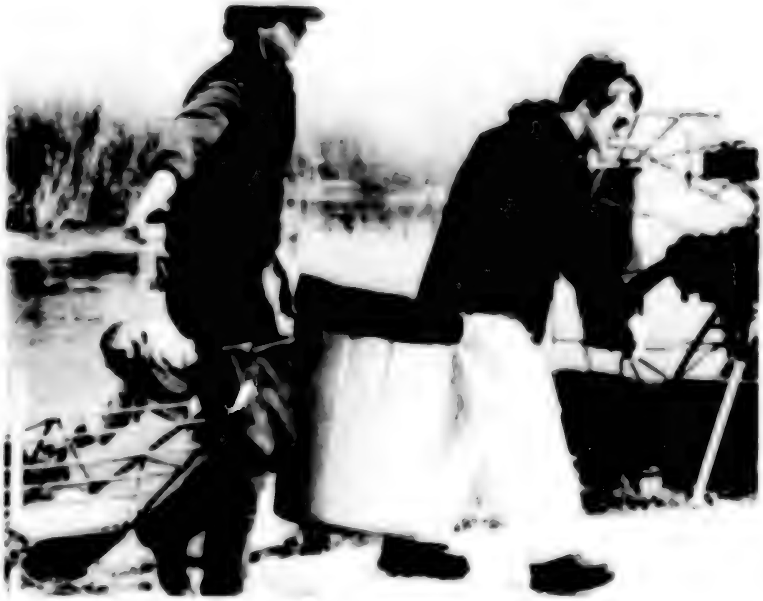
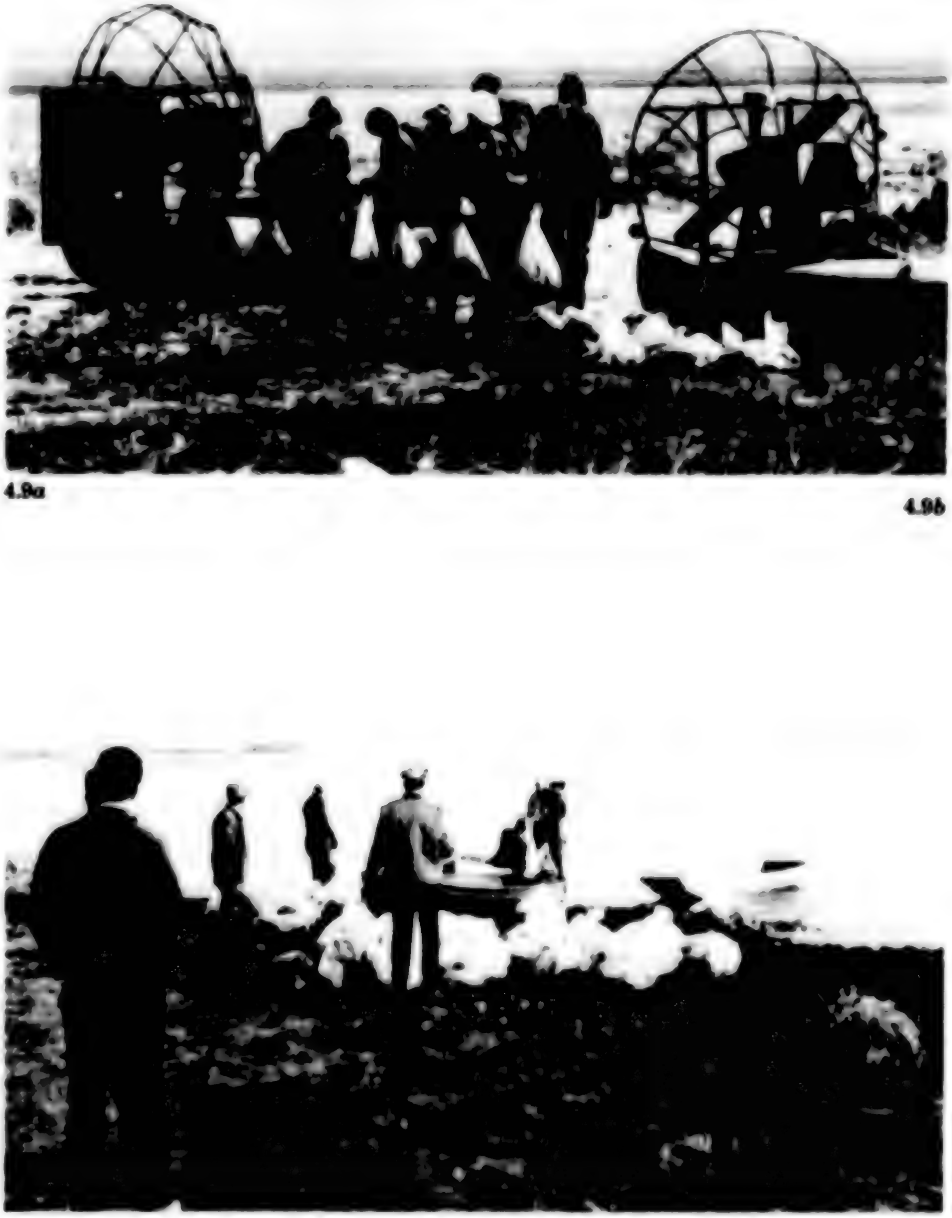


Fig. 4.8a. Plastic barrel being used to transport carcasses from collection sites by airboat to disposal site. Note use of plastic bag to line barrel. The plastic bag containing carcasses can be secured, removed, and placed in a second plastic bag for further transportation if the disposal site is not at the boat docking location, thereby allowing immediate reuse of the barrel. If the barrel containing carcasses is to be transported to some other location, the plastic bag should be tied closed and a cover placed on the barrel and secured. *M.F.*

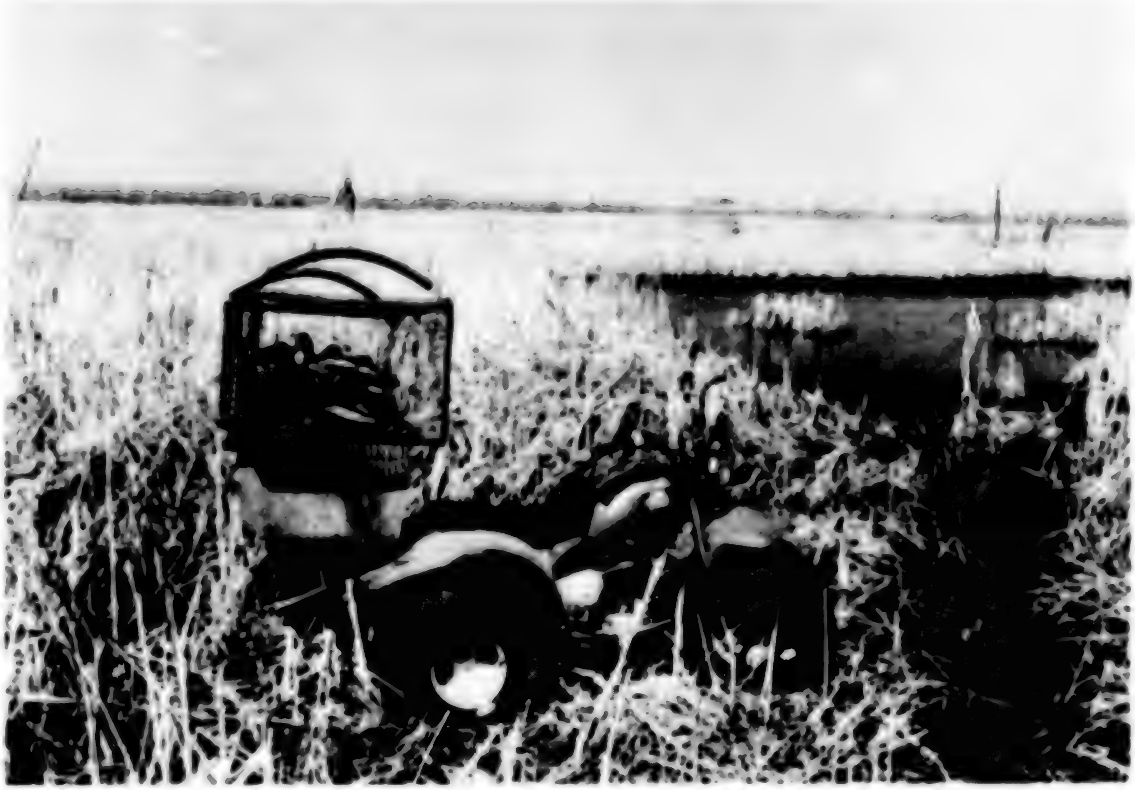
Fig. 4.8b. Example of improper transportation of carcasses to disposal site. Note untied bags, unbagged carcasses, wooden truck bed, and lack of tailgate. Carcasses and fluids contaminated with disease organisms could easily be released from the bags during transit. Fluids could contaminate the truck bed and leak to the ground through the cracks between the wooden boards. Wood absorbs fluids and is much more difficult to decontaminate than a nonporous surface. Also, carcasses could fall out of the truck because there is no tailgate. *M.F.*



4.9a

4.9b

Fig. 4.9. Different types of (*a*) motorized and (*b*) nonmotorized watercraft are useful for carcass collections. Note the use of plastic bags for containment of carcasses and further transportation to disposal sites. (*a*) *J.R.*; (*b*) *M.F.*



4.10a

4.10b

Fig. 4.10. Selection of all-terrain vehicles should be matched to local conditions. All-terrain vehicles such as (*a*) these three-wheel machines can be equipped with small baskets to hold carcasses or live birds and (*b*) be used in water no more than 2 feet deep. (*c*) Large tracked vehicles such as this equipment negotiate marshy terrain but are not amphibious. The major advantages of this equipment are the large capacity for carrying personnel, supplies, and equipment and the excellent visibility afforded by the height of the vehicle. (*d*) Small amphibious vehicles such as this six-wheel machine are capable of transporting two persons and are more stable and versatile than three-wheel vehicles but are much slower on land surfaces. (*a*) *R.W.*; (*b,c,d*) *M.F.*

4.10c

4.10d

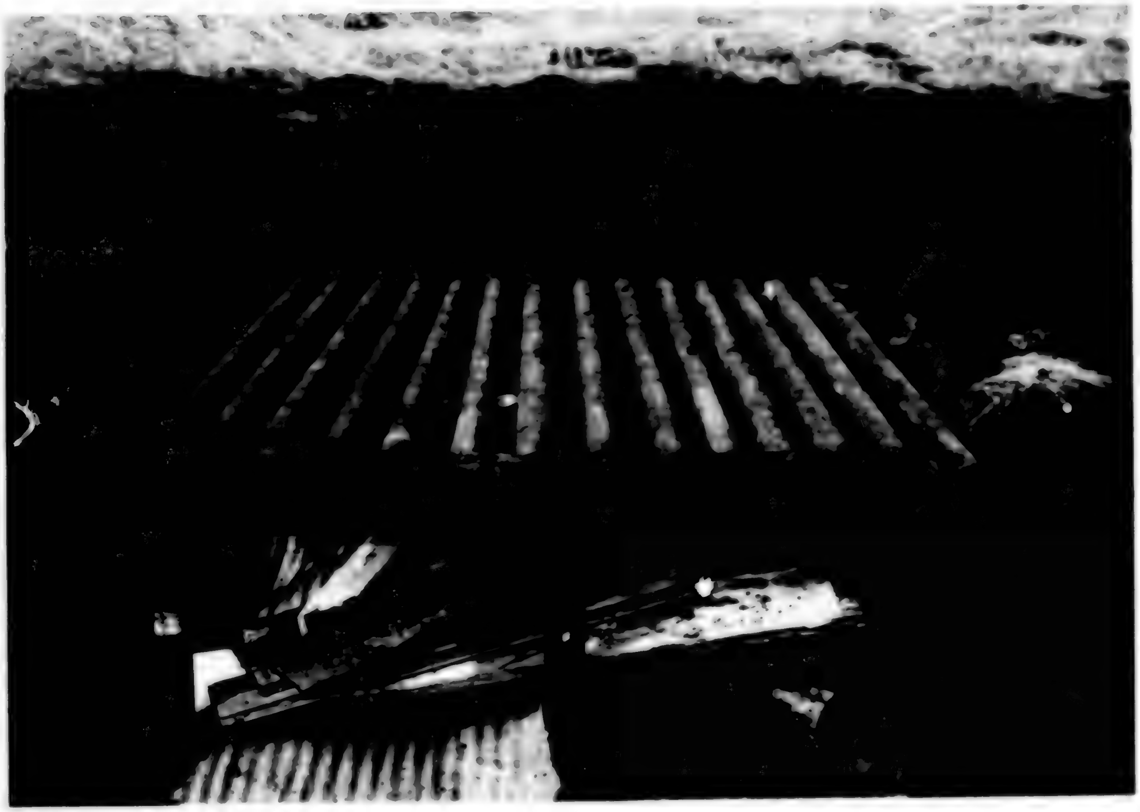
4.11*a*

4.11*b*

Fig. 4.11. Examples of portable incinerators used for disease control operations. (*a*) Garbage incinerator borrowed from State park to dispose of carcasses during Lake Andes duck plague die-off. (*b* and *c*) Locally designed and constructed incinerators in use during disease control operations. All of these are fueled with propane gas. (*a* and *c*) *M.F.*; (*b*) *C.B.*

Fig. 4.11.c

Fig. 4.12. Examples of above-ground and in-trench methods for incineration of carcasses. (*a*) Portable grate the width of a pickup truck bed fashioned from metal pipes; (*b*) simple grate suspended over pit into which carcass remains are placed for burial; (*c*) major burning pit for large-scale operation—note surrounding area cleaned of vegetation for fire protection, the size and depth of pit, burning platform, rubber tires for fuel; (*d*) intensity of heat generated by fire resulting in the bending of support pipes of the burning platform and metal grate. (*e*) Simple but sturdy above-ground structure of cinder blocks and steel grates elevated enough for fuel to be placed under the carcasses and for air to circulate upward; (*f* and *g*) highly efficient above-ground burning platform constructed of a frame of used grader blades, wire mat platform, and (*h*) sheet metal heat deflector positioned at the rear of the platform; (*i*) proper application of fuel oil (never use gasoline) for carcass incineration. Note that length of applicator prevents flashback or wind shift from endangering person applying fuel. *M.F.*



4.12*a*

4.12*b*

4.12c

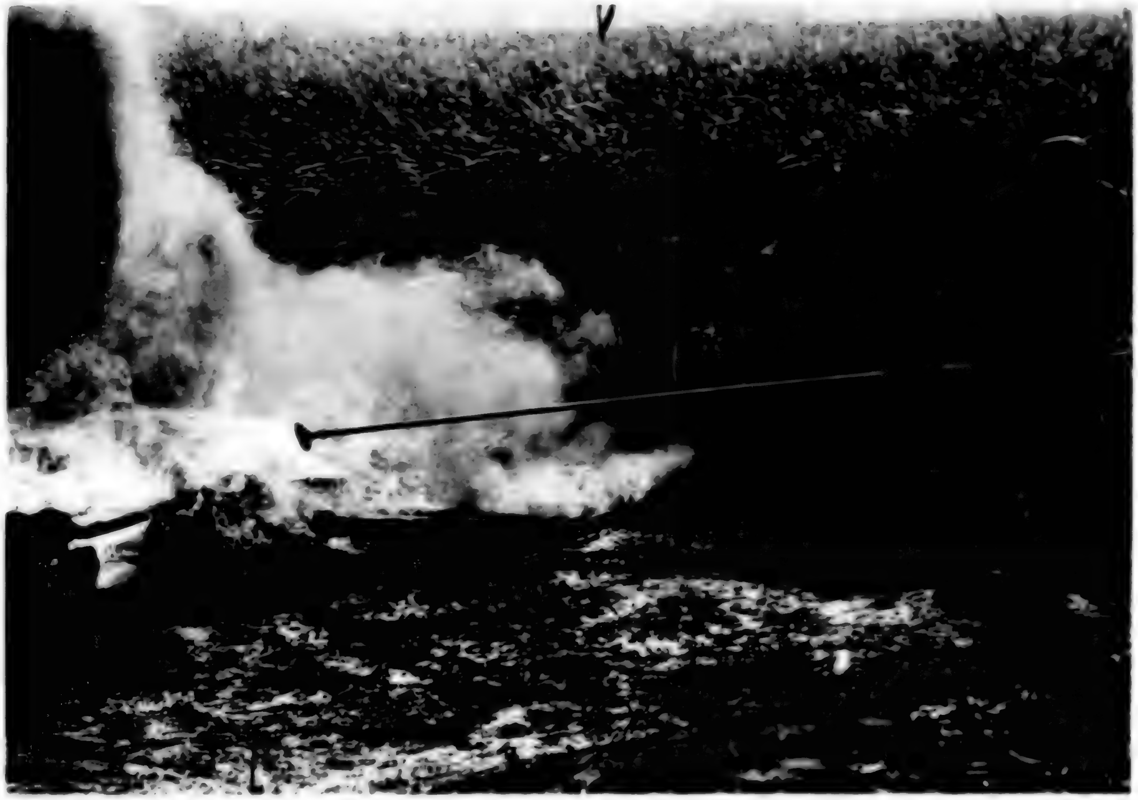
4.12d

4.12e

4.12f

4.12*g*

4.12*h*



4.12i

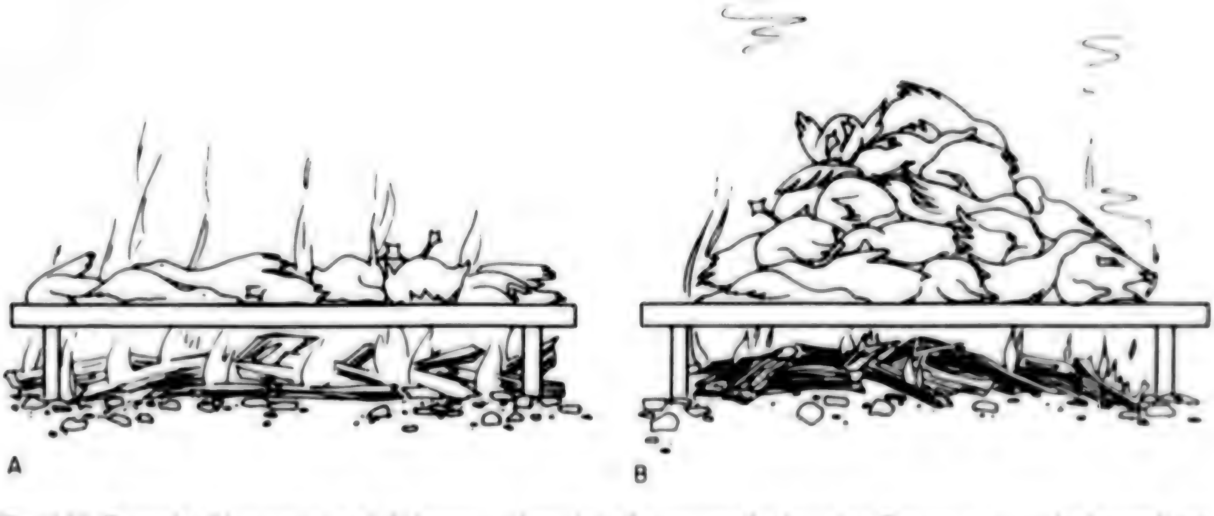


Fig. 4.13. Example of (*a*) correct and (*b*) incorrect layering of carcasses for burning. Carcas[illegible] be burned one layer at a time to prevent charred outer carcasses from insulating inner carcasses from incineration.

Animal Relocation

It is often as necessary to deal with the live, apparently healthy population during disease control activities as it is to remove and dispose of animals dying from disease. Depending on individual circumstances, the field manager may consider denying animal use of specific sites by dispersing animals from the problem area, concentrating and holding animals within a specific area, or trapping animals for sampling purposes.

Scare devices such as propane exploders (Fig. 4.14*a* and *b*) and cracker shells may be useful in keeping animals away from a toxin or infectious agent within a specific area. Hazing with airplanes, helicopters, airboats, snowmobiles, and other motorized equipment has also been successful in moving wildlife away from disease problem areas. Conversely, methods to concentrate animals for depopulation or hold them on areas to prevent their movement to problems areas include broadcasting and dumping large amounts of grain and other feed, knocking down standing grain to make it more available to wildlife, providing water through pumping operations and diversion of water flow, and providing refuge through closing the area to hunting and other interactions between wildlife and man (Fig. 4.15). Care must be taken to assure that grain used for attracting wildlife is not moldy.

4.14*a*

Fig. 4.14. Wildlife can be discouraged from use of areas by (*a*) propane exploders that function by the ignition of propane gas within the "cannon" due to the striking of a flint at a timed interval. With the exception of placing cannon and maintaining a fuel supply, this activity does not require the presence of personnel. (*b*) Manual firing of cracker shells has also been used successfully to discourage wildlife use of areas. These fireworks-like shells, should only be fired through a break-open type shotgun so that the barrel can be checked between shots to assure there are no obstructions remaining in the barrels. These shells should not be used where they can fall into dry vegetation because of the fire hazard. (*a*) *J.R.*; (*b*) *M.F.*

4.14*b*

4.15a

4.15b

Fig. 4.15. Closure of areas is often needed to assist disease control operations. *(a)* Sign used to close Lake Andes National Wildlife Refuge during duck plague die-off. *(b)* Sign used to provide refuge area to minimize bird movement during another South Dakota disease control operation. *M.F.*

Food and water are also helpful in trapping animals for assessing disease control activities. Once animals have been lured to a site, they may be captured by such devices as drugs incorporated within feed, rocket nets, drop nets, walk-in and swim-in traps, or other means of preventing escape (Fig. 4.16).

A timely response to disease outbreaks can be facilitated if such factors as need for special permits, area closures, possible involvement of endangered species, and water purchase can be anticipated and addressed before an urgent situation arises.

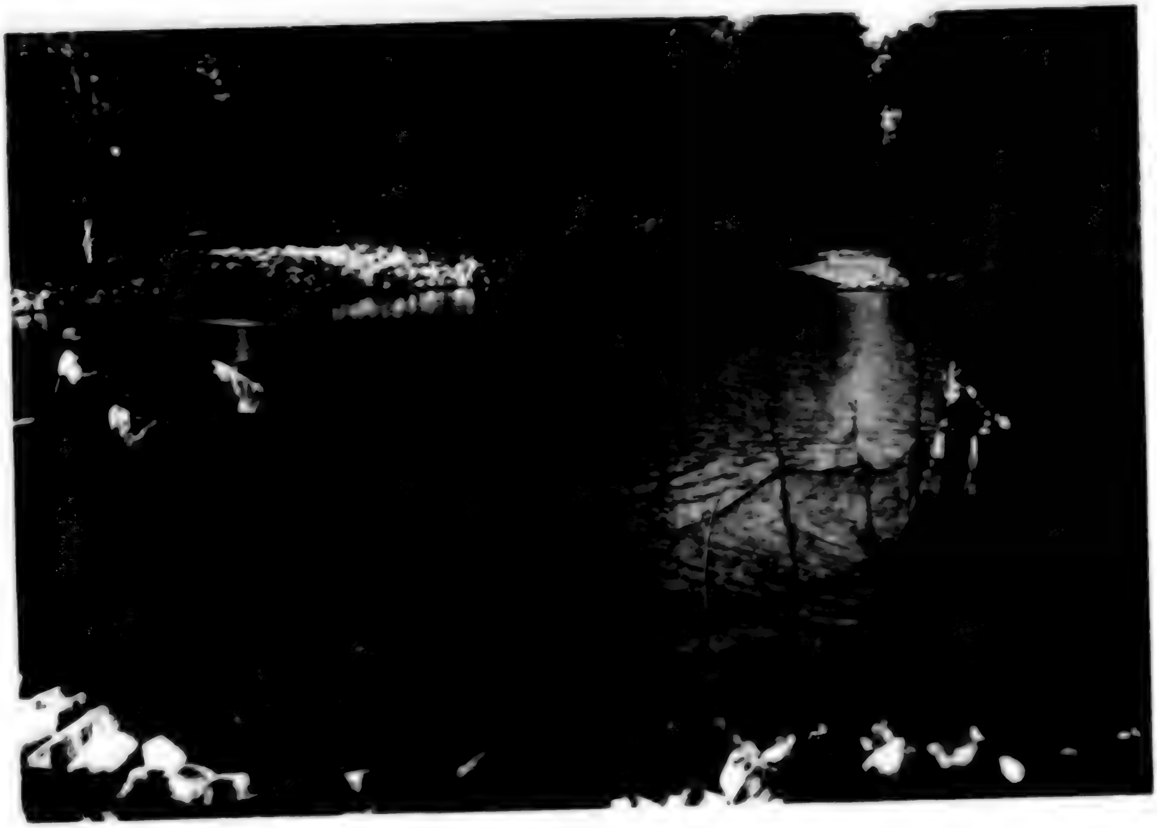
Because of the potential complexity of biological interactions in animal relocation, field managers should seek the advice of disease control specialists whenever possible before taking independent action. As a general rule, animal dispersal is not recommended when infectious disease is involved, unless it can be assured that the population being dispersed will not infect other wildlife. Also, it is important that water manipulation not produce conditions favorable to development of botulism or other disease problems.

Fig. 4.16. Various types of capture devices are useful for disease operations. *(a)* Rocket net being fired over Canada geese; *(b)* snow geese captured by cannon nets; *(c)* constructing a funnel trap to capture birds in a zoological park; *(d)* capturing birds within a funnel trap; *(e)* capturing flightless Canada geese in drive trap; *(f)* capturing waterfowl in large, baited funnel trap; *(g)* using drugged grain to capture birds in residential situations. When drugs are used, maintain close surveillance of the situation so that animals that become drugged, such as the bird *(h)* lying on its back, can be promptly collected before they are seized by other animals or drown if they venture into the water before the drug takes effect. *M.F.*

(over)

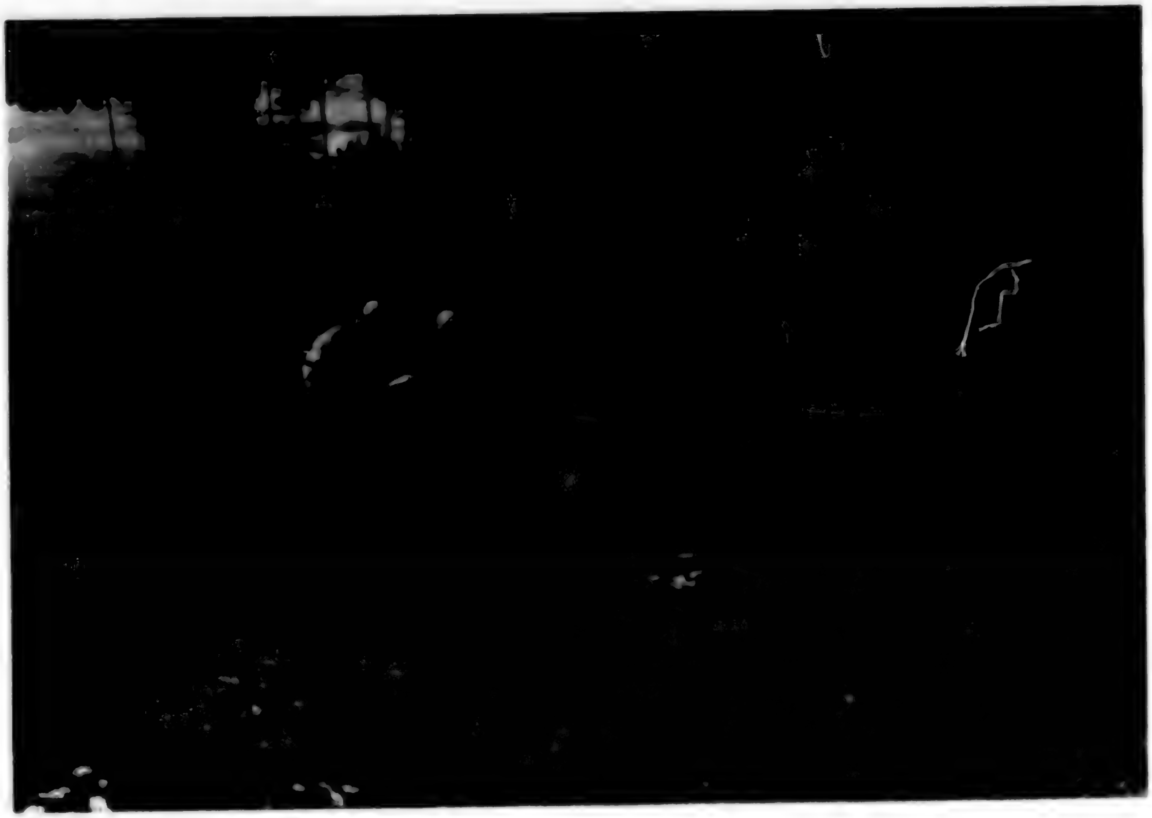
4.16*a*

4.16*b*



4.16c

4.16*d*



4.16e

4.16f

4.16g

4.16h

Disinfection

The purpose of disinfection is to prevent the mechanical transmission of disease agents from one location to another by people, equipment, and supplies. Some viruses, bacteria, and other infectious agents have considerable environmental persistence. This may require disinfecting the local environment involved in a disease outbreak to prevent recurrence of the disease when the site is used by other animals.

Clothing worn during disease control operations should be thoroughly washed (coveralls and clothes worn under protective raingear) before being used again. Personnel should shower and shampoo their hair before leaving the site, if possible, but always before contact with other wildlife areas. Boots should always be disinfected before personnel enter vehicles when they are in contaminated areas, and all equipment should be disinfected to the extent possible before it is moved from the area (Fig. 4.17*a* and *b*). Special attention should be given to the underside of vehicles (Fig. 4.17*c* and *d*). Motor vehicles should be put through a car wash before they are allowed on other areas, and boats and all-terrain vehicles should receive a thorough washing and cleaning before leaving the area.

Always conduct disinfection of a disease outbreak site under the direct guidance of disease control specialists. Large-volume tanks, and pumps that can be operated from mobile units such as trucks (Fig. 4.18) and boats, are especially useful in these situations.

Disinfection procedures require a suitable disinfectant, containers for the disinfectant after it has been diluted to appropriate strength, and a way of applying the disinfectant. Environ (Vestal Laboratories, St. Louis, Missouri 63110; telephone [314] 535-1810) is a highly effective, biodegradable disinfectant that comes in two strengths (Environ and 1-Stroke Environ). Refuge managers and other field managers should consider keeping a supply of this disinfectant on hand for general use. 1-Stroke Environ should be diluted 1:256. Chlorine bleach (e.g., Clorox) is also a highly suitable disinfectant and is readily available at most grocery stores. For general use this should be diluted with water 1:10. Stronger concentrations (1:5) should be used when disinfecting heavily contaminated areas.

Stiff bristle brushes, buckets and containers that can be used for foot baths, and pressure or hand sprayers that can be used to dispense the disinfectant are also needed. Here again, field managers should not stockpile materials beyond their normal needs. Instead, the station contingency disease plan should identify readily available sources of these supplies and equipment.

When the disease problem involves an infectious agent, personnel handling contaminated materials should refrain from working with similar species or those susceptible to the disease for at least 7 days after completion of their disease control activities. For example, a field manager involved in an intensive avian cholera disease control operation on Monday should not be banding waterfowl on that refuge or elsewhere on Friday of that week.

Personnel

Labor-intensive operations such as carcass removal and disposal sometimes require more personnel than are usually employed on an area. In other instances, specialized help such as surveillance by low-level aircraft may be needed. Two major considerations in deciding to use nonstation personnel for routine operations are the station manager's ability to maintain control of nonstation personnel actions and the potential for the die-off to be educational to outside personnel. For example, the use of local sportsman clubs to help with carcass collections during a major lead-poisoning die-off can be highly effective in changing any negative attitudes they might have toward nontoxic shot use on the area.

Sportsman clubs, retriever clubs, biology and wildlife classes at local universities and colleges, local chapters of conservation organizations such as the Audubon Society, the active military and

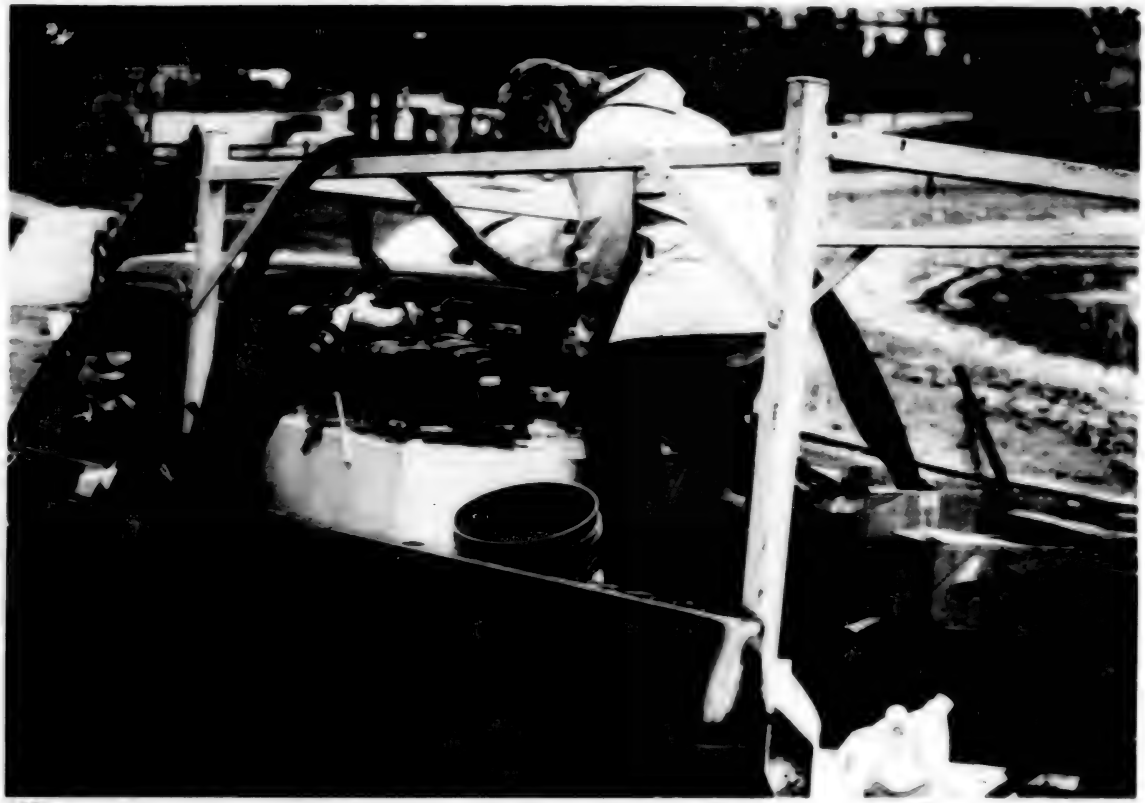
Fig. 4.17. Equipment and personnel should be disinfected to the extent possible before leaving disease operation areas. (*a*) Initial disinfection procedures should take place well within the contaminated area. (*b*) Boots and other items in contact with the ground should receive a second application of disinfectant at the point where entry is made into the "clean area" as is being done at the location where the specimen chest is being transferred. (*c* and *d*) Various types of spray units can be used to apply disinfectant to the underside of vehicles. Tires and wheel wells are the primary areas of concern as they may contain contaminated soil or animal fecal material from the disease area. *M F*

4.17a

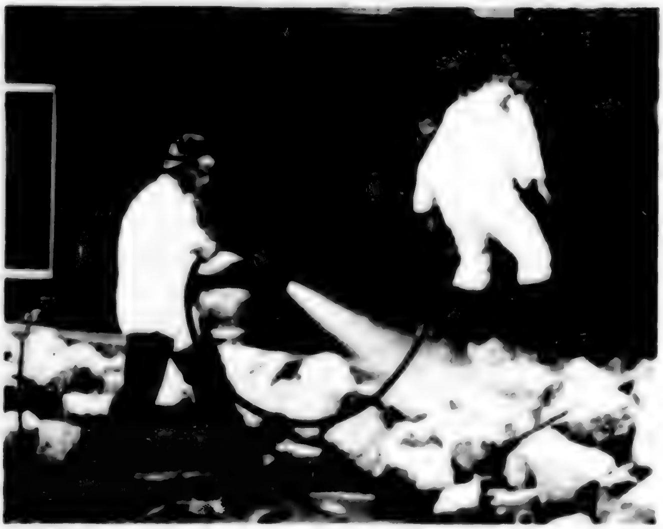
4.17b

4.17*c*

4.17*d*



4.18*a*



4.18*b*

Fig. 4.18. (*a*) Portable tank and pump mounted on truck bed for dispersing disinfectant during duck plague control operation and (*b*) application of that disinfectant to a structure used to house birds. The long length of hose on this unit allowed all areas of major bird use to be reached from service and perimeter roads. (*a*) *T.A.*; (*b*) *M.F.*

National Guard (also of value for technical assistance), and similar groups have all provided volunteer assistance in combating disease problems at various times and places. Sound judgment must be exercised in selecting and using volunteers because of legal liability in case of an accident. Nevertheless, do not overlook this source of assistance. Contingency plans should list groups and organizations and contact persons for each, their telephone numbers, and an approximation of the work force and times of availability (e.g., weekends only or Wednesday only). For technical assistance, list the specific type of activity (e.g., bulldozer operator or helicopter pilot).

In addition to preparing a contingency plan, wildlife personnel should become familiar with the other phases of disease control operations. Table 4.1 provides a descriptive outline of these phases. Especially relevant to field managers are the equipment and supply needs identified under the Disease Response Section of Table 4.3.

Disease control operations can be seriously undermined without current assessment of wildlife morbidity and mortality, and the cause of disease problems. When infectious or highly toxic agents are involved, detecting disease problems early is critical in preventing the problem from becoming widespread. Also, failure to accurately assess the cause of the die-off can result in control actions actually contributing to the magnitude of losses and spread of the problem. Different types of disease problems require different types of response. One cannot assume that the current die-off is due to the same cause as previous die-offs that have occurred on the area or that only one disease agent is responsible. It is not uncommon for two or more causes of wildlife mortality to occur on an area simultaneously. Control of these different diseases may call for opposite types of actions, thereby requiring a more comprehensive strategy to be developed for the disease control operation.

Refuge managers and other field biologists greatly influence the effectiveness of disease control operations by their responsiveness, their knowledge of the local situation, how well they are prepared, the flexibility they maintain, their resourcefulness, and when possible, their ability to obtain appropriate technical assistance and training for combating disease problems. Timely and properly carried-out disease control activities can significantly reduce the magnitude of wildlife losses that might otherwise occur.

Table 4.1. *Outline of disease control operations.*

I. Planning

A. *Identify Needs*

1. Sources of additional personnel to help during disease emergencies. Potentially, these include
    a. state and federal agencies
    b. active military and National Guard
    c. private conservation agencies
    d. local sportsmen clubs
    e. local universities
2. Sources and availability of equipment and supplies for disease control operations (Appendix C)
3. Special needs
    a. burning permits
    b. Section 7 consultations
    c. lodging and meal facilities for work crews
    d. ability to attract and hold animals on site-specific areas by providing food, water, refuge, or other means
    e. ability to deny animal use of specific areas by scaring devices and other means
    f. ability to capture animals for sampling, immunization, or other needs

B. *Record Biological Information*

1. Daily and seasonal animal movement patterns within the general area
2. Migration patterns and population peaks for major and endangered species
3. Past history of diseases

C. *Prepare Contingency Plan (See Tables 4.2 and 4.3)*

II. Intitial Response

A. *Identify Problems*

1. Obtain diagnosis by submitting carcasses to a qualified diagnostic laboratory as soon as mortality or morbidity is evident. (See Chapters 3 and 4 for shipping procedures)
2. Conduct field investigation to determine extent of problem (i.e., species, number of animals, and geographic area involved).
3. Identify special biological, biopolitical, or physical considerations associated with problem. Before proceeding further with II. B and C, the advice of a specialist.

B. *Establish Control of Area*

1. Close affected area, when warranted, to all but authorized personnel.
2. Identify special work areas for disease control activities.
    a. carcass disposal sites
    b. laboratory investigations area
    c. briefing area for news media and staff
    d. vehicle parking
    e. assembly areas for arriving workers
    f. command post
3. Initiate carcass clean up, but do not dispose of carcasses without guidance from disease control specialists.

C. *Communications*

Notify appropriate agency and nonagency personnel of die-off.

Table 4.1. *Outline of disease control operations. (continued)*

III. Disease Control

A. *Response*

1. Disease control actions are dictated by the type of disease, environmental factors, species involved, and other circumstances. Typically, actions associated with major die-offs require:
   - a. bringing personnel, equipment, and supplies on-site
   - b. organizing work force, briefing workers about the problem, and assigning duties
   - c. carcass pickup and disposal
   - d. monitoring cause of mortality to detect changes in the cause of the problem (die-offs often involve more than a single cause and different control actions may be required for these different causes)
   - e. decontamination of personnel and equipment
   - f. news media briefing sessions and "show-me" trips[a]

B. *Management*

1. Disease management activities often involve:
   - a. population manipulation such as removal, controlled movement including relocation and local concentration of animal populations, and population dispersal
   - b. habitat manipulation to prevent, attract, or maintain animal use of an area

2. Decontamination of the infected environment, such as:
   - a. chemical treatment of land, water, and structures
   - b. vegetation and water removal (desiccation) to allow air and sunlight (ultraviolet) to destroy microorganisms
   - c. controlled burning to remove vegetation and dispose of mechanical structures

IV. Surveillance

A. *Monitoring*
After disease control operations have ended, the area should be kept under surveillance for 10 to 30 days to watch for additional flare-ups.

B. *Investigations*
This stage is also an appropriate time to conduct follow-up investigations of factors that helped cause and sustain the problem, and to carry out animal population and environmental sampling to discern disease exposure patterns and environmental reservoirs of disease agents.

V. Analysis

Each disease control operation provides a learning experience. It is important to the success of future operations to evaluate what was done, the degree of success achieved, problems encountered, and what should have been done differently.

[a] Media briefing sessions and "show-me" trips should be conducted by personnel having comprehensive knowledge of the situation.

Table 4.2. *Equipment and supplies used in disease control operations.*

Activity	Equipment and supplies
A. *Carcass Collection*	
1. Transportation of personnel	a. all-terrain and four-wheel vehicles, snowmobiles b. airboats, canoes, other boats c. helicopter d. waders, snowshoes
2. Transportation of carcasses	a. large, heavy-duty plastic bags b. plastic trash cans with lids c. sleighs and trailers d. trucks, boats e. strapping tape and other means of securing closure of containers
B. *Carcass Disposal*	
1. Burial	a. earth-moving equipment for digging trenches or pits (bulldozer, backhoe) b. shovels c. lime or fuel oil to spread on carcasses d. any applicable permits
2. Incineration	a. portable incinerators and fuel b. local permanent incinerator c. earth-moving equipment for digging trenches or pits (bulldozer, backhoe) d. burning permits e. shovels f. metal grates and cinder blocks for building burning platforms g. sheet metal or metal roofing for heat reflectors h. fuel for burning carcasses (wood, coal, rubber tires, fuel oil, napalm) i. fire suppression equipment
C. *Sanitation Procedures*	
1. Decontamination of environment and structures	a. chemicals such as Environ, chlorine bleach b. pumps and suction apparatus for drainage of water areas c. buckets, brushes d. spray application by aircraft, power systems mounted in trucks and boats, and hand-carried spray units
2. Protection of personnel and prevention of mechanical movement of disease agents to secondary locations by people and equipment	a. raingear, coveralls, rubber gloves, rubber foot gear, hats b. spray units and chemical disinfectants such as Environ and chlorine bleach c. plastic bags for transportation of field clothes to laundry d. brushes, buckets e. disposable gloves, hats, coveralls, and foot coverings

Table 4.2. *Equipment and supplies used in disease control operations. (continued)*

Activity	Equipment and supplies
D. *Field Communications*	
1. Field activities	a. portable radios for communication between field personnel b. radios in vehicles for communication between field units and between units and command post
2. Information activities	a. typewriter for preparing briefing documents b. maps, acetate, and other supplies for overlays depicting die-off and control activity information c. telephone lines for communication with others, supply operations d. transportation for news media "show-me" trips
E. *Surveillance and Observation*	
1. Field activities	a. aircraft and pilots certified for low-level flights (500 feet and below) for monitoring animal populations and environmental conditions b. binoculars and spotting scopes
2. Office activities	a. maps, acetate, and other supplies for tracking the progress of events and animal populations associated with die-off b. telephone for contacting others to trace movement of migrant animal populations that might enter problem area or that have departed problem area
F. *Animal Population and Habitat Manipulation*	
1. Denying animal use of a specific area	a. aircraft, boats, snowmobiles, and other motorized means of hazing animal populations b. propane exploders c. crackers shell, break-open shotguns, and protective face shield d. audio systems and other scare devices e. pumps for draining water or adding water to areas
2. Concentration and maintenance of animals in a specific area	a. grain and other sources of food b. pumps and water to provide habitat c. "No Hunting" and "Area Closed" signs to provide temporary refuge area
G. *Animal Sampling and Monitoring*	
1. Animal capture	a. cannon nets and other capture equipment b. grain and other baits to lure animals to capture site
2. Animal marking	a. visible marking devices such as paint, neck collars, and other devices b. permanent marking devices such as leg bands and ear tags c. temporary marking devices such as radio transmitters

Table 4.3. *Station disease contingency plan.*

Introduction

I. Size, configuration, and other important characteristics of station area conveyed with help of tables, maps, photographs, station brochures, public use maps, and similar documents

II. Record of previous disease outbreaks, including nature of disease, species involved, magnitude of die-off, and season and year (Table 4.4)

Disease Surveillance

I. Brief outline of current surveillance activities on station and adjacent areas—State, Federal, and private

II. Identify disease reporting and notification procedures (names, titles, organization, and telephone numbers of persons to be contacted)

Disease Response

I. *Logistical Considerations*

A. Personnel Sources (telephone numbers, addresses, names of contact persons)
   1. Local, State, and Federal agencies (military, university)
   2. Sportsman clubs and volunteers

B. Equipment (types and numbers on-site, and sources off-site)
   1. Vehicles (conventional and all-terrain)
   2. Aircraft (fixed-wing and rotary)
   3. Earth-moving equipment (backhoe, bulldozer)
   4. Pumps (for flooding or draining marshes)
   5. Boats (motor, self-propelled, air boats)
   6. Radios (portable and fixed); during nonfire seasons the Boise Interagency Fire Center, 3905 Vista Avenue, Boise, Idaho 83704, is a potential source for obtaining assistance for very large communication needs
   7. Incinerators
   8. Decontamination units (sprayers)
   9. Scaring devices (propane exploders, sirens)
   10. Freezers
   11. Portable toilets (construction site type)

C. Supply Sources (identify sources, addresses, and telephone numbers of local or closest sources)
   1. Disinfectants and chemicals
   2. Plastic bags
   3. Fuel for carcass burning
   4. Field clothes (gloves, rainwear, coveralls, boots)
   5. Plastic trash barrels, tubs, scrub brushes
   6. Scaring devices (cracker shells, fireworks); provide contact telephone number and address for local animal damage control office
   7. Dry ice and liquid nitrogen
   8. Grain and other wildlife foods
   9. Nearest shipping address for air and ground receipt of goods and supplies

D. Lodging for Temporary Personnel Assigned to Disease Control Operation

E. Food
   1. On-site capabilities
   2. Off-site capabilities (give consideration to early and late hours)

Table 4.3. *Station disease contingency plan. (continued)*

- F. Identify Working Areas (diagrams are sufficient; limited narrative may also be required; Fig. 4.2)
  - 1. Clean areas
    - a. command post (must have adequate telephones)
    - b. news media briefing room
    - c. parking
    - d. eating areas
    - e. staff assembly and rest areas
    - f. equipment and supply receipt
    - g. other
  - 2. Transition areas
    - a. decontamination of personnel
    - b. decontamination of equipment
  - 3. Contaminated areas
    - a. carcass disposal
    - b. laboratory investigations
    - c. animal holding

II. *Biological Considerations* (Provide data in charts, figures, photographs, maps, tables)

- A. Species and Population Data
  Major species (identify by season of presence, relative abundance, and peak population periods)

- B. Animal Movement Patterns (Figs. 4.3 through 4.5)
  - 1. Daily
  - 2. Seasonal
  - 3. Production and dispersal patterns

- C. Weather Patterns
  - 1. Freeze-up and ice-out periods
  - 2. Major periods of precipitation and drought
  - 3. Other (temperature profiles, major periods of haze, fog, and high winds)

- D. Habitat and Population Manipulation Potential
  - 1. Methods (water manipulation capability, feeding)
  - 2. Anticipated population response to habitat (movement, concentration, dispersal)

III. *Communications* (Provide lists of principal local and regional contact personnel and telephone numbers)

- A. State Agencies
  - 1. Conservation
  - 2. Agriculture
  - 3. Health Department
  - 4. University diagnostic laboratories

- B. Federal Agencies
  - 1. Environmental Protection Agency
  - 2. U.S. Department of Agriculture
  - 3. Public Health Service

- C. Other Organizations
  - 1. Cooperating organizations (e.g., area representatives of Audubon Society, National Wildlife Federation, Ducks Unlimited)
  - 2. Local sportsman clubs
  - 3. Private wildlife area managers
  - 4. Local game breeder organizations
  - 5. Local domestic animal husbandry and production operations

Table 4.3. *Station disease contingency plan. (continued)*

	D. Media 1. Television 2. Radio 3. Newspapers
Supplemental Information	I. Location of Nearby Laboratories (hospitals, universities, county and State facilities). II. Federal and State Permit Status for Biological Collections. III. Burning Permits IV. Regulatory Requirements V. Background Information (e.g. water sources, water quality data, potential sources of disease transmission between wildlife and domestic animal concentrations.) VI. Identification and Location of Adjacent or Nearby Wildlife Refuges, Management Areas, and Private Preserves. VII. Identification of Unusual or Politically Sensitive Aspects of Area.

Table 4.4. *Example of a disease outbreak summary, Mosquito Flat National Wildlife Refuge.*[a]

Disease	Date	Location	Principal species involved	Estimated population at risk[b]	Losses		Control efforts	Diagnostic laboratory
					Carcass count	Estimated total		
Unknown	Aug–Oct 1972	Unit 6 and Yellowleg Flat on adjacent State land	Northern pintail Teal	5,000	1,500	3,000	None	None
Lead poisoning	April 1980	Mud Lake	Redhead Tundra swan	–	75	200	None	State diagnostic laboratory
Lead poisoning	Dec 1983	Mud Lake	Mallard Canada goose	10,000	–	150	Blinds were relocated in 1984	Veterinary Science Dept., State college
Unknown	Jan 1983	Mud Lake	Muskrat	3,000	100	500	None	Veterinary Science Dept., State college
Avian botulism	July–Sept 1985	Units 3,4,5	Shorebirds Northern pintail Teal	25,000	2,200	10,000	Drained Unit 4, flooded 3 and 5	National Wildlife Health Center

[a] The material in this table is fictitious and for illustration only.

[b] Number of animals using the area involved in the die-off.

Chapter 5.
Euthanasia

Patricia A. Gullett

Euthanasia: An easy or painless death

Background

In the course of a disease outbreak, sick or even healthy animals may be caught, killed, and sent to a diagnostic laboratory. It is everyone's responsibility to see that these deaths are carried out by the most humane method available. Humane death is possible only when immediate insensitivity to pain is achieved by depression of the central nervous system (CNS). The CNS includes the brain and spinal cord.

There is no one perfect euthanasia technique appropriate to all situations. In choosing a technique, consider the following factors:

- Does it present a risk to human health and safety?
- Does it induce immediate CNS depression?
- Does it require stressful capture and restraint?
- Is it appropriate for this size or type of animal?
- Will it interfere with diagnostic tests?
- Is specialized equipment or training required?
- Will this technique offend the casual observer?
- Is it time- and cost-effective?

Euthanasia methods may be chemical or physical. Physical euthanasia mechanically destroys the CNS. Chemical euthanasia introduces a toxic agent into the body by injection or inhalation.

Physical euthanasia may be induced by:

- Cervical dislocation
- Decapitation
- Stunning and exsanguination (bleeding out)
- Shooting

Chemical euthanasia may be induced by:

- Lethal injection of an anesthetic or toxic agent
- Inhalation of an anesthetic gas
- Inhalation of a toxic gas

Physical Euthanasia

Cervical Dislocation

Immediate CNS depression occurs when the brain is separated from the spine. The separation must take place at the base of the brain, or within the upper-third of the neck (cervical spine). Separation is induced in small- to medium-sized birds and in small mammals by grasping the base of the skull in one hand and the body (usually at the base of the neck) in the other hand. Pulling rapidly and firmly in opposite directions will separate the cord (Fig. 5.1*a*).

In duck-sized birds, grasp the base of the skull and swing or twirl the bird in a circular, snapping motion three or four times (Fig. 5.1*b*). Be sure to feel the separation of bones and cord but *do not attempt this technique without training!* This technique is effective, quick, inexpensive, requires no special equipment, and only minimally affects diagnostic testing.

Cervical dislocation may upset the casual observer because animals, especially birds, convulse for several seconds to minutes after death. These movements are due to spinal reflexes and pain is not involved.

Decapitation

Severing the head from the body, like cervical dislocation, induces immediate CNS depression. It is especially useful for geese, swans, or other birds too large for cervical dislocation. A heavy knife, machete, hatchet, or bolt cutters should be used to ensure that the spinal cord, encased in the neck bones, is severed. Emasculatomes, available from veterinary supply houses, crush the cord without cutting, thereby reducing blood loss. The same uncontrolled movement seen after cervical dislocation will follow decapitation. Protect yourself, as always, from contamination as the blood may contain toxic or infectious agents.

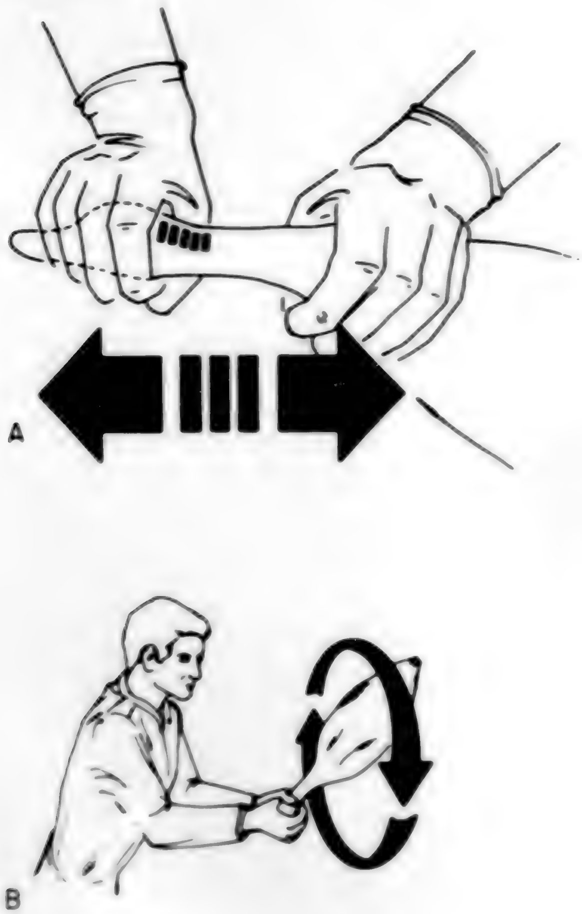


Fig. 5.1. Cervical dislocation procedures. (*a*) The brain can be separated from the spine in small- to medium-sized animals by grasping the animal at the base of the skull with one hand, at the base of the neck with the other, and pulling rapidly and firmly in opposite directions with a strong snapping action. (*b*) For larger birds (duck and goose size), the spinal cord can be more easily separated by grasping the bird at the base of the skull and swinging or twirling the bird in a circular, snapping motion (see text for additional information).

Stunning and Exsanguination (Bleeding Out)

A *forceful* blow to the center of the skull will produce massive brain hemorrhage and immediate CNS depression in birds and small mammals. To ensure death, cut the major blood vessels in the neck and allow the animal to bleed out. This technique should not be used if the brain is required for diagnostic tests such as rabies, or if the euthanasia must be performed in public.

Shooting

Central nervous system depression and death can be induced by shooting an animal at close range in the brain or high in the cervical spinal cord. The type and caliber of firearm is dependent on the size of the animal to be killed. The brain is the preferred site for euthanasia, but the cervical spine may be used if the brain is required for diagnostic tests. Training and experience are required to ensure a humane death and also to reduce the human safety hazards.

Chemical Euthanasia

To administer lethal injections, personnel must be trained in injection techniques as well as in the safe handling and disposal of needles, syringes, and drugs.

Lethal Injection

Special preparations are available which, when injected into the blood, cause immediate CNS depression followed shortly by a cessation of breathing and heartbeat.

Use of these preparations for wildlife euthanasia is limited. They produce an aesthetically acceptable euthanasia, but the animal must first be captured and restrained, causing anxiety to the animal and risking injury to the handler. Dangerous animals should be sedated or stunned first to allow for proper administration of the drug. If large animals (> 100 pounds) or many animals must be killed, lethal injection can be time-consuming and expensive.

Sleepaway (Ft. Dodge Laboratories, Inc., Ft. Dodge, Iowa) and Beuthanasia-D Special (Burns-Biotic Laboratories, Inc., Omaha, Nebraska) are concentrated barbiturate solutions plus additives. The solutions are inexpensive but, due to the potential for human abuse, require licensing by the Federal Drug Enforcement Administration (DEA) for purchase, use, and storage. The DEA requires considerable record-keeping. T-61 Euthanasia Solution (American Hoechst Corp., Somerville, New Jersey) does not require DEA licensing. This advantage is countered by its increased cost. The effective dosage and administration technique for T-61 are printed on the product label. All of these solutions are toxic if accidentally or intentionally self-administered.

Lethal injections may not be appropriate in certain instances since drug residues interfere with some tests. Check first with the diagnostic laboratory to see if the proposed euthanasia technique is compatible with the testing to be performed.

Strychnine or paralytic drugs such as nicotine sulfate or succinylcholine are not approved for use as euthanasia agents.

Anesthetic Gas

Inhalation of certain products (halothane, methoxyflurane, and nitrous oxide) are routinely used in clinical medicine to produce and maintain anesthesia. Ether and chloroform, used routinely in the past, have been replaced by these safer agents. Ether is explosive and chloroform is toxic to humans. An overdose of any of these agents is fatal.

Halothane is the gas of choice. Methoxyflurane vaporizes slowly and therefore has a longer anesthetic induction time. Nitrous oxide has a low potency and is available only in gas form (others are purchased as a liquid, vaporizing at room temperature and normal air pressure). There is limited field application for these agents. Animals that are difficult to handle may be rendered unconscious with the gas, then killed with another method. For example, a bear in a culvert trap may be exposed to a gas to induce anesthesia. The bear could then be euthanized with a lethal injection. Smaller animals can be euthanized with an overdose of gas only.

An animal exposed to anesthetic gas may pass through an "excitation phase" before becoming unconscious. The animal may vocalize and appear to struggle for a short time. This behavior may be distressing to the casual observer and can be dangerous for the animal handler. Reptiles and some marine mammals may hold their breath when exposed to the gas and might not receive an effective dose.

It is important to be sure that the animal is dead—not just unconscious—before shipment, necropsy, or disposition. After removal from the gas environment, the animal may wake up quickly, with little warning. Remember this when working with raptors or carnivores.

To administer the gas, confine the animal in a cage or crate and cover with plastic. Pump in gas or soak a cloth with the liquid and place it inside the plastic. Cold temperatures will decrease the rate at which the liquid becomes gas. Wait at least 15 minutes before checking for death.

Since all of these gases constitute a human health hazard, including the potential for causing spontaneous abortion and congenital abnormalities, the workplace must be wellventilated.

Toxic Gas

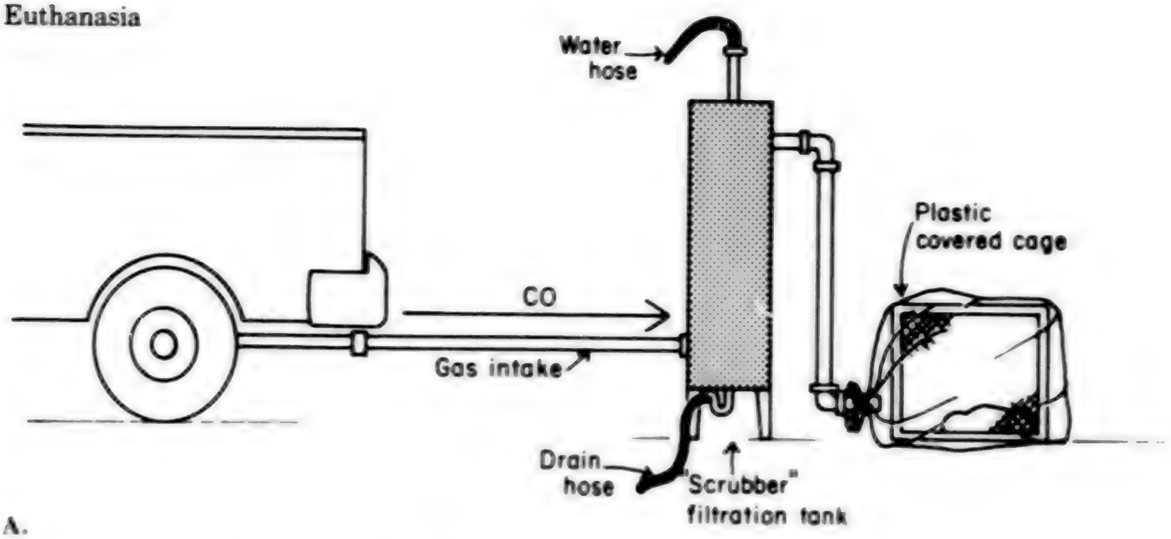
Toxic gas such as carbon monoxide (CO) or carbon dioxide (CO_2) may be useful when many small animals must be killed. It must be borne in mind, however, that a 1% concentration of carbon monoxide is lethal and represents a substantial human safety hazard. As with anesthetic gas, all work with this gas must be conducted in an open area.

The least expensive source of carbon monoxide is an internal combustion engine. An automotive four-cycle engine at idle speed works best. Two-cycle engines should not be used, and smog-control devices must be bypassed. This is most easily done by disconnecting the exhaust system from the manifold at the engine and attaching the hose to the exhaust system at this point. Adjusting the carburetor to burn richer will increase the amount of CO. The preferred fuel is unleaded regular gasoline without additives. The engine should not be burning oil and should be warmed up before use.

To avoid discomfort to the animals, the carbon monoxide must be cooled and filtered before use. It may be bubbled through water or, preferably, through a filter "scrubber," which can be built. Fig. 5.2 is taken from *Carbon Monoxide Chamber, Suggestions on Construction and Operating Instruction* published by the American Humane Association. This model can be adapted for field use if the principles of cooling and filtering are maintained.

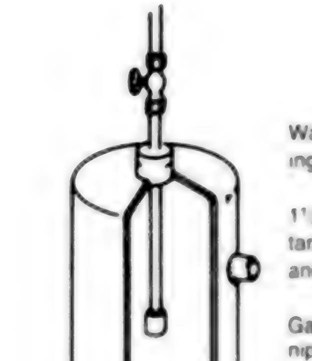
Carbon dioxide may be purchased in cylinder, or it may be obtained in smaller quantities from CO_2 fire extinguishers or in solid state (dry ice). Animals must be protected from contact with the dry ice. The gas should be warmed to ambient temperature before exposing animals. This is accomplished by passing it through hoses surrounded by warm water. This gas is heavier than air. A deep layer must be built up in the killing chamber so the animal will be unable to reach higher, more oxygenated air (Fig. 5.3).

Cages covered with plastic bags or plastic garbage cans can be used as killing chambers. Leave the animal in the chamber until breathing and heartbeat have ceased.



A.

Fig. 5.2. (*a*) Example of how to use carbon monoxide for euthanasia of animals. (*b*) Diagram of "scrubber" tank (see text for additional information).



Water spray to be on continuously during operation of chamber

1¼" galvanized half coupling welded on tank end with 1¼" × ⅜" brass bushing and ⅜" × 12" brass nipple

Gas outlet to CO chamber 2" galv. half nipple welded on, thread outside

Water spray ⅜" brass spray nozzle, full circle type

Course gravel - (not limestone) Size: will screen past 2" mesh and retained by ¾" mesh

Gas intake from engine. 2" × 20" Galvanized nipple inlet welded in, thread end outside tank

B. Carbon monoxide gas "scrubber" and filter

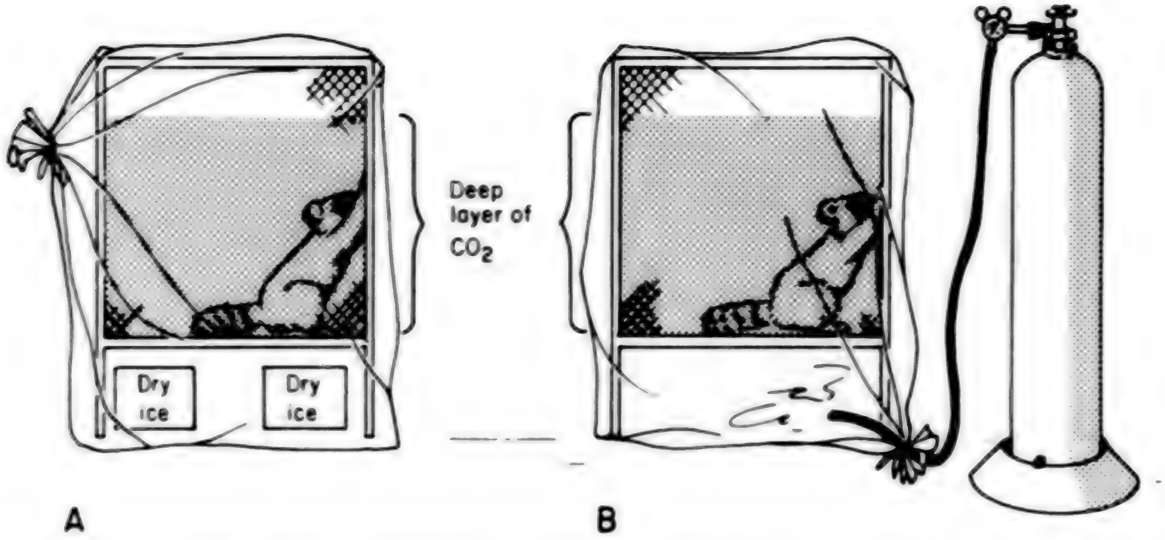


Fig. 5.3. Use of carbon dioxide for euthanasia of animals. *(a)* Evaporation of dry ice within a plastic-shrouded cage. *(b)* Direct application of carbon dioxide in a gas form. Because this gas is heavier than air, a deep layer of gas must be built up so that the animals being euthanized cannot get above the gas. The chamber containing the animals must not be airtight or gas buildup may result in an explosion. Openings should be at the top of the chamber.

Checklist

Before performing any euthanasia technique, think through the following list:

- How many animals must I collect?
- Is the euthanasia technique appropriate to the animal, the diagnostic requirements, the possible audience?
- Do I have all necessary equipment and assistance assembled for euthanasia?
- Do I have all necessary equipment assembled for collection of blood, tissues, or culture from the fresh-dead animal?
- Do I have all necessary equipment assembled for chilling, storage, or shipment of the carcass or tissues?

After completing euthanasia, be certain that specimens being collected are properly identified, preserved, and packaged for transportation to the diagnostic laboratory (see chapter on Specimen Collection and Preservation, and Specimen Shipment). Be sure to indicate the euthanasia technique used.

Supplementary Reading

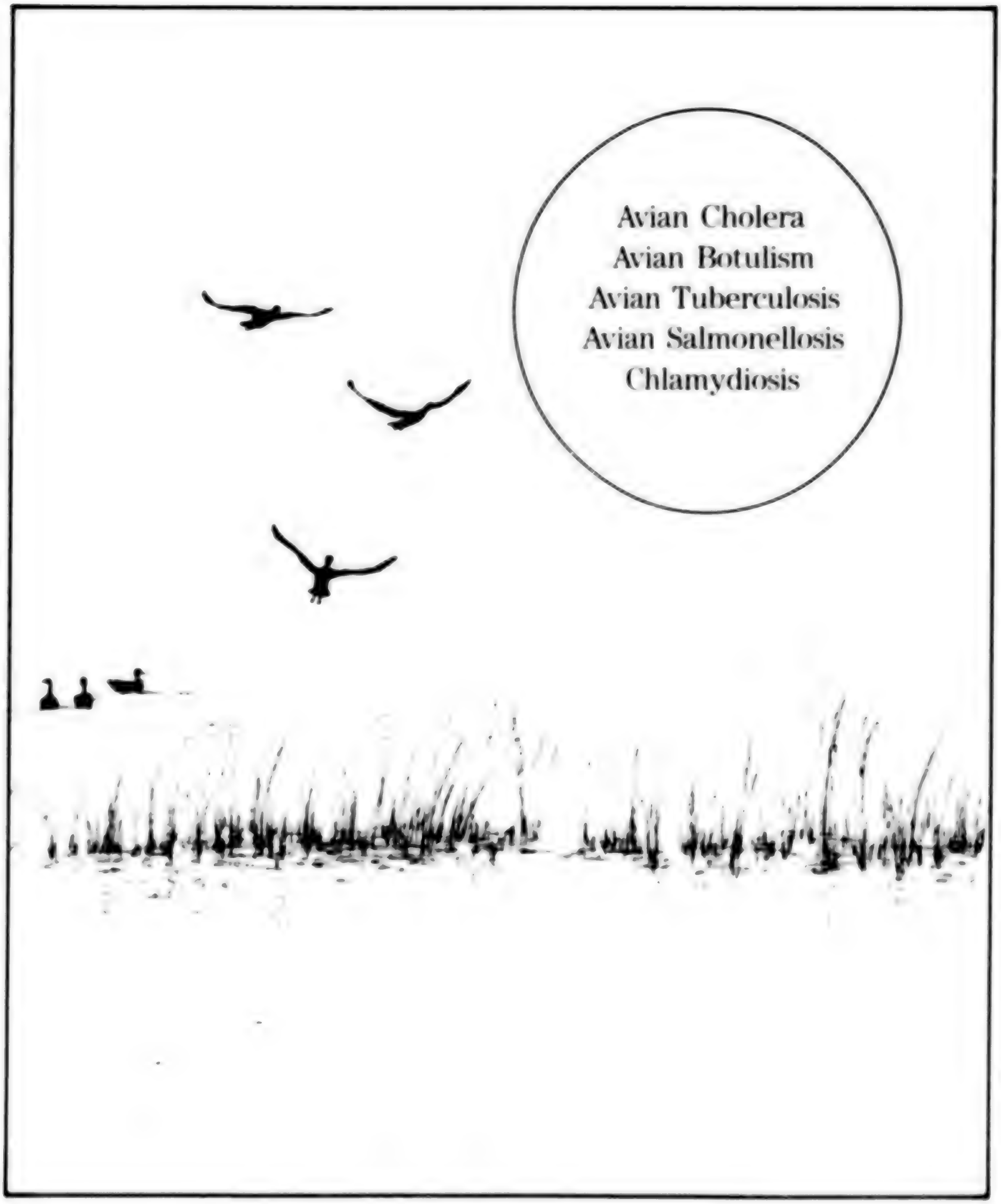
American Humane Association. Carbon monoxide chamber: suggestion on construction and operating instructions. Information series. The American Humane Association, P.O. Box 1266, Denver, Colo. 80201.

Smith, A. W., chairman. 1986 Report of the American Veterinary Medical Association panel on euthanasia. 1 February 1986. J. Am. Vet. Med. Assoc. 188:253–268.

Section 2.

Diseases of Migratory Birds

Bacterial Diseases



Avian Cholera
Avian Botulism
Avian Tuberculosis
Avian Salmonellosis
Chlamydiosis

Chapter 6.
Avian Cholera

Milton Friend

Synonyms

Fowl cholera, avian pasteurellosis

Cause

Avian cholera is a highly infectious disease caused by the bacterium, *Pasteurella multocida*. Acute infections are common and can result in death 6 to 12 hours after exposure. Under these circumstances "explosive" die-offs involving more than 1,000 birds per day have occurred in wild waterfowl. More chronic infections with longer incubation times and less dramatic losses also occur. Transmission can occur by bird-to-bird contact, ingestion of contaminated food or water, and perhaps in aerosol form.

Species Affected

It is likely that most species of birds and mammals can become infected with *P. multocida* (Fig. 6.1). However, *P. multocida* is a heterogenous organism. The ability of different strains of this bacterium to cause clinical disease in different animal species is highly variable. Most (if not all) bird species are susceptible to clinical disease following exposure to virulent strains of *P. multocida* commonly found in waterfowl. All isolates from avian cholera outbreaks have been found to readily kill laboratory mice. Specific relationships between bird and mammal strains of this bacterium are not well understood. Strains isolated from cattle have not been shown to readily cause clinical disease in birds.

Scavenger species such as crows and gulls are commonly diagnosed as having died from this disease, but deaths of raptors such as hawks and eagles from avian cholera are far less frequent (Fig. 6.2). Major identified outbreaks in waterfowl in any year are but a small percentage of actual losses. Species losses for most major outbreaks are closely related to species composition and abundance during the period of the die-off (Fig. 6.3).

Distribution

Avian cholera was unreported in free-living migratory birds in the United States before 1944. Losses have now been reported coast-to-coast and border-to-border. The occurrence of this disease within the United States has increased dramatically since 1970, and avian cholera now ranks with avian botulism and lead poisoning as major causes of waterfowl mortality. The frequency and severity of avian cholera outbreaks vary greatly among areas (Fig. 6.4). This disease has also been diagnosed in waterfowl in many countries, including Canada, but not Mexico. This is probably due to lack of surveillance and reporting rather than to absence of this disease in Mexico.

In the United States there are four major focal points for avian cholera in waterfowl: the Central Valley of California; the Tulare Lake and Klamath Basins of northern California and southern Oregon; the Texas Panhandle; and Nebraska's Rainwater Basin. The movement of avian cholera from these areas follows the well-defined pathways of waterfowl movement. Spread of this disease along the Missouri and Mississippi river drainages is also consistent with waterfowl movement. No consistent patterns of avian cholera outbreaks exist within the Atlantic Flyway except for periodic occurrences in eider ducks nesting off the coast of Maine (Fig. 6.5).

Fig. 6.1. Partial list of species from which *Pasteurella multocida* have been isolated.

Mammals

- Domestic animals
  - Cattle, horses, swine, goats, sheep
  - Dogs, cats
  - Gerbils, rabbits

- Big game
  - Elk and deer
  - Caribou and reindeer
  - Bighorn sheep
  - Bison

- Carnivores
  - Bear
  - Lynx, bobcat, puma
  - Foxes
  - Weasel, mink
  - Raccoon

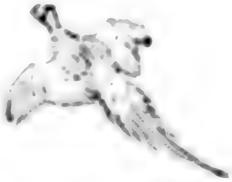
- Rodents
  - Rats, mice, voles
  - Muskrat, nutria
  - Chipmunk

Birds

- Domestic poultry
  - Chickens, turkeys
  - Ducks, geese
  - Pigeons

- Water birds
  - Ducks, geese, swans
  - Cranes
  - Pelicans
  - Herons
  - Gulls
  - Grebes

- Raptors
  - Hawks, owls, eagles
- Upland game birds
  - Pheasants



- Other
  - Crow
  - Songbirds

Fig. 6.2. Relative occurrence of avian cholera in wild birds.

Type of bird	Frequency
Waterfowl	● ● ●
Coots	● ●
Wading birds	○
Cranes	●
Shorebirds	●
Gulls	● ●
Raptors	○
Upland game birds	○
Crows	● ●
Songbirds	●

● ● ● Common occurrence, major die-offs occur almost yearly

● ● Frequent occurrence including occasional major die-offs

● Small number of reports generally involving individual or small numbers of birds

○ Infrequent, rare, or not reported

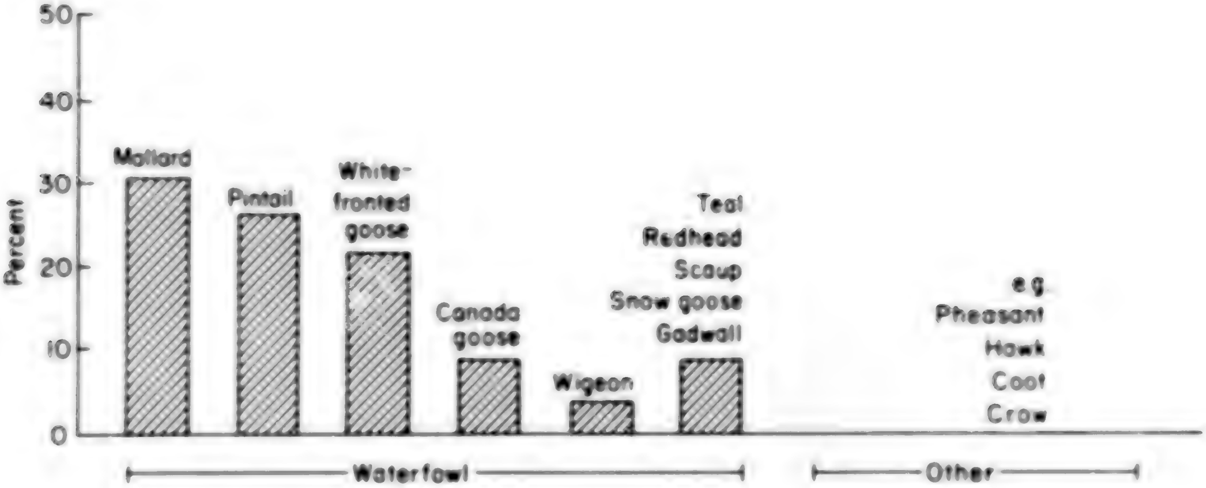


Fig. 6.3. Relative occurrence of mortality from avian cholera in Nebraska's Rainwater Basin during spring 1980.

Seasonality

Losses can occur at any time of the year (Fig. 6.6). However, a predictably seasonal pattern exists in areas where avian cholera has become well established as a disease of wild waterfowl. A major loss of snow geese occurred on Canadian breeding grounds in addition to losses of breeding eiders in Maine and Quebec. Outbreaks in California normally start during fall and continue into spring. Late winter is the peak time for avian cholera in the Texas Panhandle, and spring migration has resulted in annual losses from this disease in Nebraska's Rainwater Basin since 1975 and in western Saskatchewan, Canada, since 1977.

Field Signs

Few sick birds are seen during avian cholera outbreaks because of the acute nature of this disease. However, the number of sick birds increases when a die-off is prolonged over several weeks. Sick birds often appear lethargic or drowsy (Fig. 6.7) and can be approached quite closely before attempting escape. When captured, these birds often die quickly, sometimes within a few seconds or minutes after being handled. Other birds have convulsions (Fig. 6.8), swim in circles, or throw their heads back between their wings and die (Fig. 6.9). These signs are similar to those seen in duck plague and in some types of pesticide poisoning. Other signs include erratic flight, such as flying upside down before plunging into the water or onto the ground and attempting to land a foot or more above the surface of the water; mucous discharge from the mouth; soiling and matting of the feathers around the vent, eyes, and bill; pasty, fawn-colored, or yellow droppings; and blood-stained droppings or nasal discharges (both also occur with duck plague).

Always suspect avian cholera when large numbers of dead waterfowl are found in a short time, few sick birds are seen, and the dead birds appear to be in good flesh. Death can be so rapid that birds may literally fall out of the sky or die while feeding, with no signs of illness. When sick birds are captured and die within a few minutes, avian cholera should also be suspected. None of the signs described above are unique to this disease; their occurrence should be recorded as part of any history being submitted with specimens and must be considered along with lesions seen at necropsy.

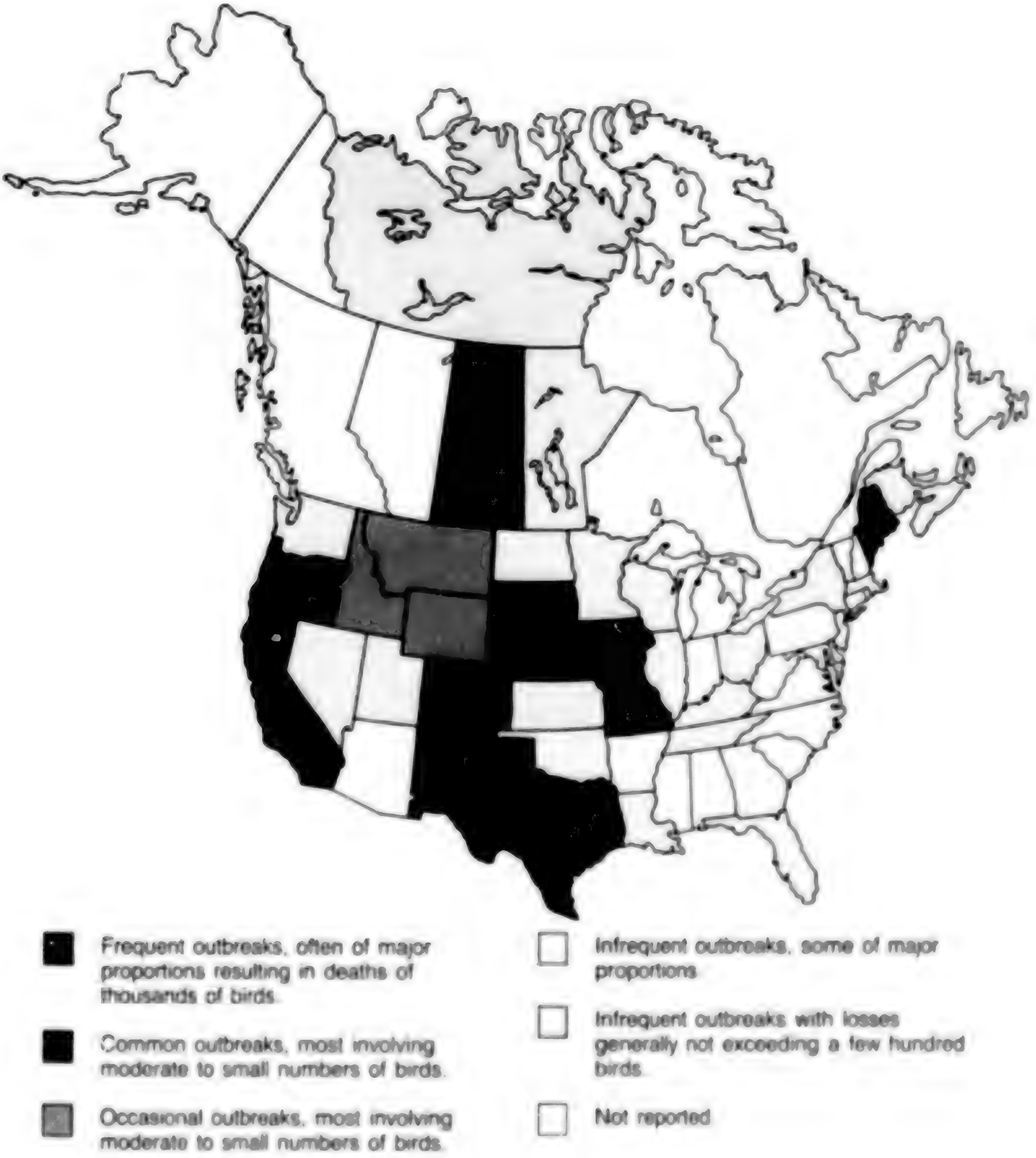

■ Frequent outbreaks, often of major proportions resulting in deaths of thousands of birds.
■ Common outbreaks, most involving moderate to small numbers of birds.
▨ Occasional outbreaks, most involving moderate to small numbers of birds.
☐ Infrequent outbreaks, some of major proportions.
☐ Infrequent outbreaks with losses generally not exceeding a few hundred birds.
☐ Not reported.

Fig. 6.4. Reported distribution of avian cholera in wild birds.

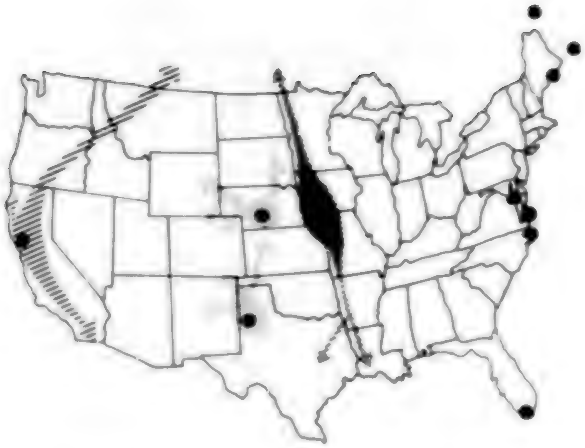


Fig. 6.5. The occurrence of avian cholera in waterfowl seems to be closely related to bird movements west of the Mississippi River. There is no apparent pattern for outbreaks along the Atlantic seaboard.

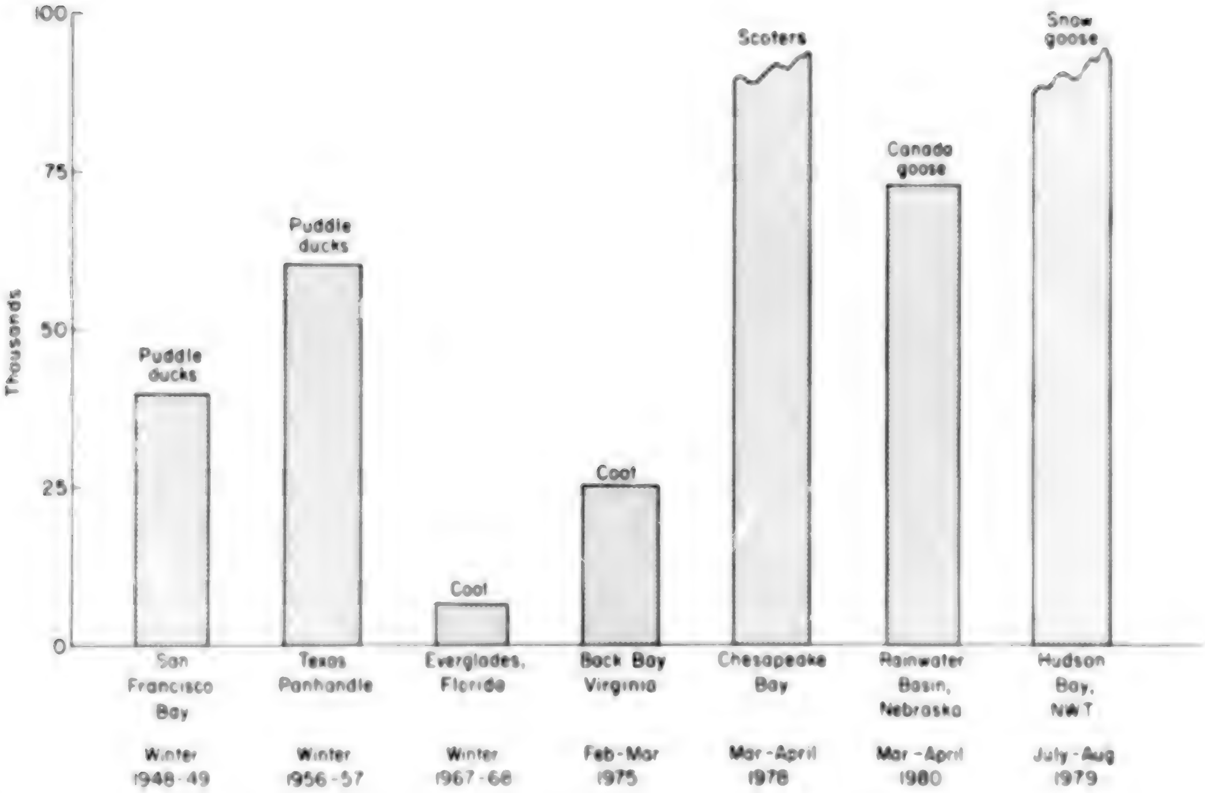


Fig. 6.6. Examples of seasonal occurrence, principal species, and geographic diversity of major avian cholera outbreaks in wild birds. (Broken bars indicate very high but indeterminate mortality.)

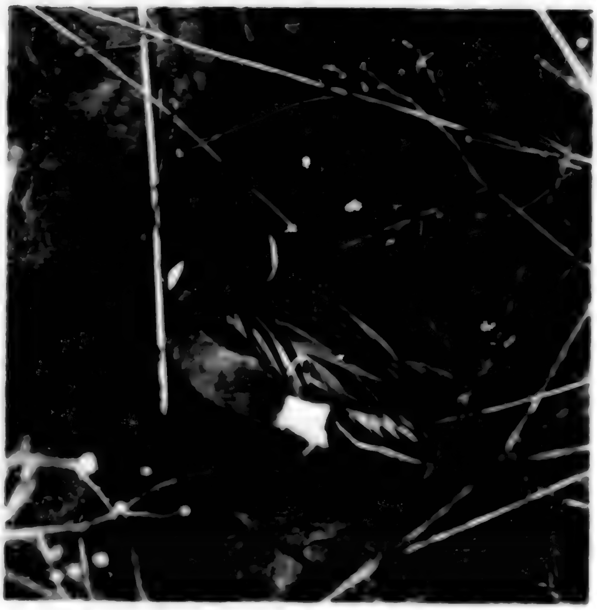


Fig. 6.7. Lethargic appearance of drake northern pintail with avian cholera. *M.F.*

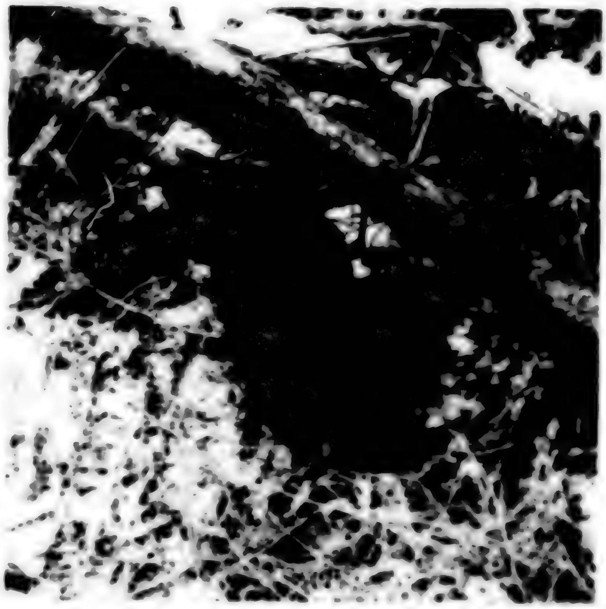


Fig. 6.8. Avian cholera-infected crow in convulsions. *M.F.*

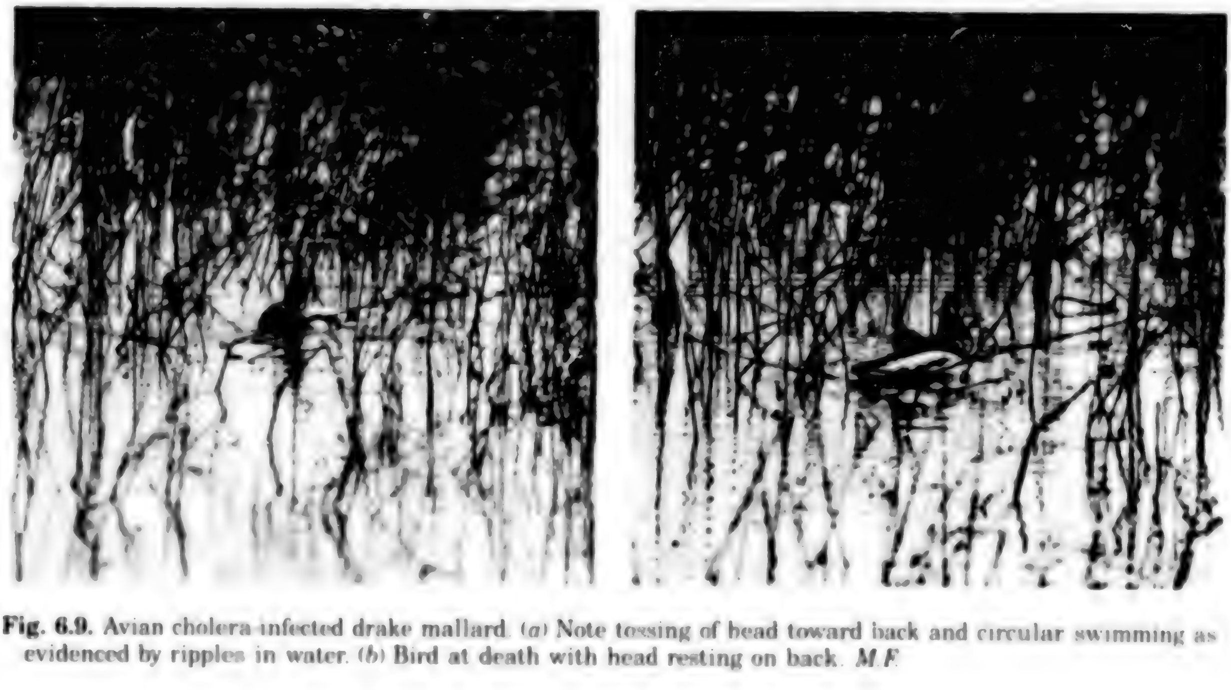


Fig. 6.9. Avian cholera-infected drake mallard. (*a*) Note tossing of head toward back and circular swimming as evidenced by ripples in water. (*b*) Bird at death with head resting on back. *M.F.*

Gross Lesions

Under most conditions, birds that have died of avian cholera have substantial amounts of subcutaneous and visceral fat (except for seasonal losses of fat). The most prominent lesions seen at necropsy involve the heart and liver and sometimes the gizzard. Hemorrhages of various sizes are frequently found on the surface of the heart muscle or the coronary band (Fig. 6.10). Hemorrhages are also sometimes visible on the surface of the gizzard. Areas of tissue death that appear as small white to yellow spots are commonly seen within the liver. Where the area of tissue death is greater, the "spots" are larger and in some instances the area of tissue death is quite extensive (Fig. 6.11).

The occurrence of the abnormalities described for heart, liver, and gizzard are dependent on how long the bird lived after it became infected. The longer the survival time, the more abundant and dramatic the lesions. In addition, there may be changes in the color, size, and texture of the liver. There is a darkening or a copper tone to the liver, and it may appear swollen and rupture when handled. Because birds infected with avian cholera often die so quickly, upper portions of the digestive tract may contain recently ingested food. All of these findings are similar to what might also be seen with duck plague (duck virus enteritis or DVE), and illustrate the need for a laboratory diagnosis.

The lower portions of the digestive tract (below the gizzard) commonly contain thickened yellowish fluid that is heavily laden with *P. multocida* (Fig. 6.12).

Diagnosis

As with all diseases, isolation of the causative agent is required for a definitive diagnosis. Submitting a whole carcass provides the diagnostician with the opportunity to evaluate gross lesions seen at necropsy and also provides all appropriate tissues for isolation of *P. multocida*.

When it is not possible to send whole carcasses, tissues should be sent that can be collected in as sterile a manner as possible in in the field. The most suitable tissues for culturing are heart blood, liver, and bone marrow. Remove the entire heart and place in a Whirl-Pak bag for shipment as identified in the chapter on Specimen Collection and Preservation; do not attempt to remove the blood from the heart. The liver should also be removed and placed in a separate bag; if it cannot be removed intact, submit a major portion of this organ (at least half). Refrigerate these samples as soon as possible after collection and insure that they are kept cool during shipment. When shipment is to be delayed for more than a day or transit time is expected to exceed 24 hours, freeze these specimens.

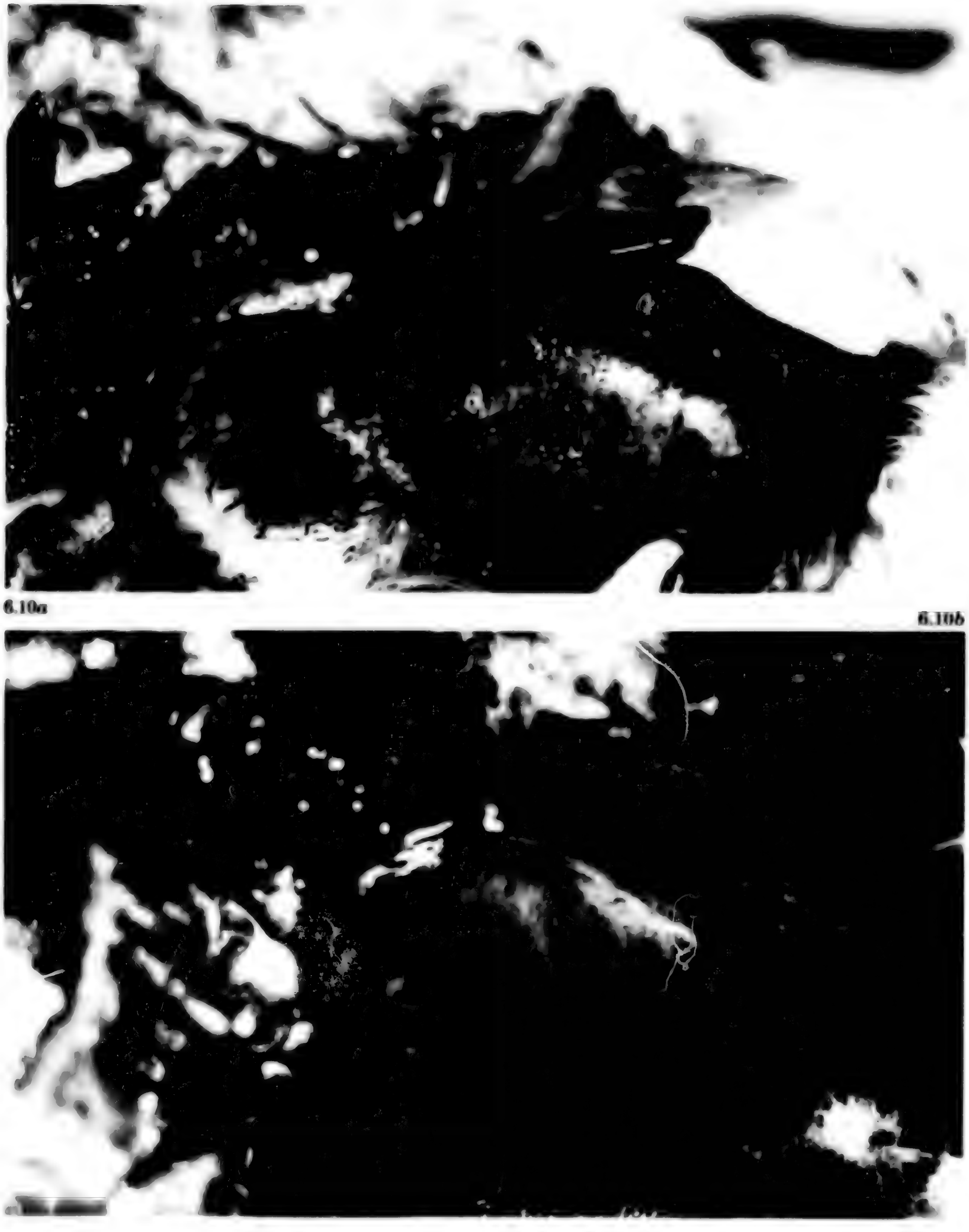
P. multocida persists for several weeks to several months in bone marrow. The wings of badly scavenged or decomposed carcasses should be submitted whenever avian cholera is suspected as the cause of death, and more suitable tissue samples are not available.

Control

Avian cholera is highly infectious and spreads rapidly through waterfowl and other migratory bird populations. This process is enhanced by the gregarious nature of most waterfowl species and by dense concentrations of migratory water birds that result from habitat limitations. Prolonged environmental persistence of this bacteria further promotes new outbreaks. Pond water remained infective for 3 weeks after dead birds were removed from one area in California; survival in soil for up to 4 months was reported in another study; and persistence of this organism in decaying bird carcasses occurred for at least 3 months.

Early detection of avian cholera outbreaks is a first line of defense in controlling this disease. Frequent surveillance of areas where migratory birds are concentrated and the timely submission of carcasses to disease diagnostic laboratories allow disease control activities to be initiated before the outbreaks reach advanced stages. The opportunity to prevent substantial losses is greatest during the early stages of outbreaks, and costs are minimal in comparison with handling a large-scale die-off. Control actions need to be focused on minimizing exposure of migratory and scavenger bird species to *P. multocida* and minimizing environmental contamination by this organism.

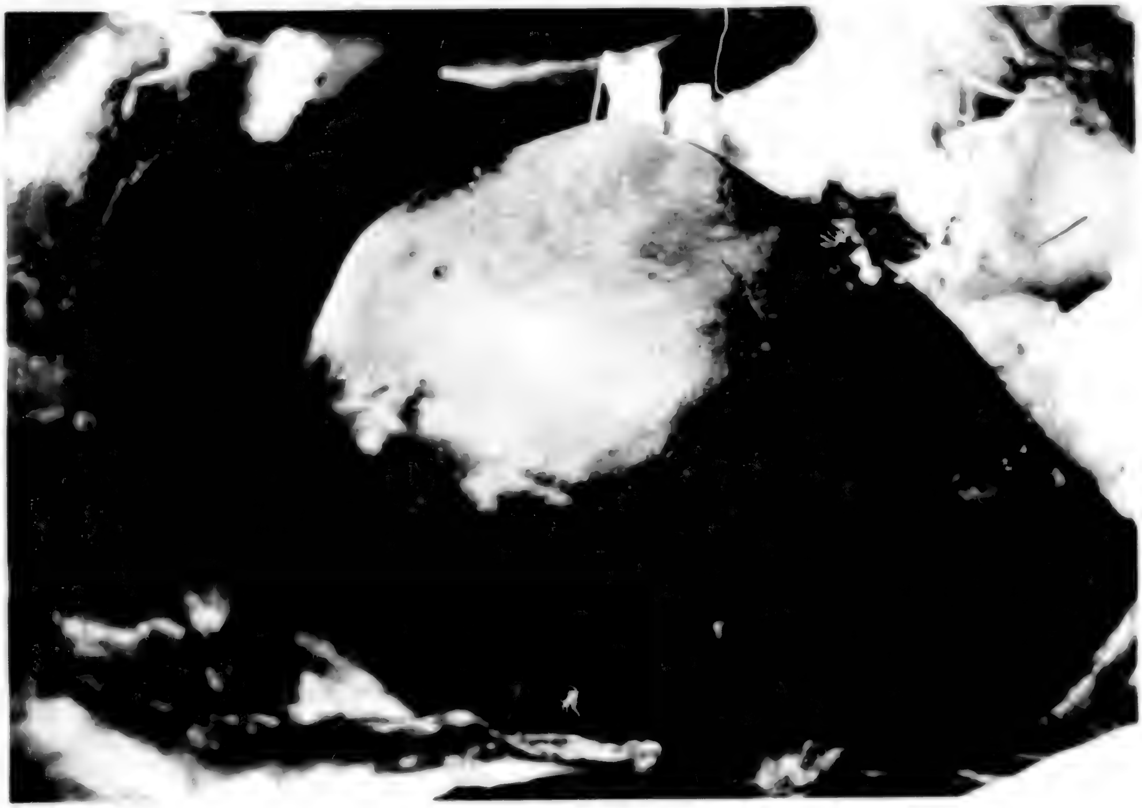
We recommend rigorous collection and incineration of carcasses as standard procedures. Carcass



6.10a

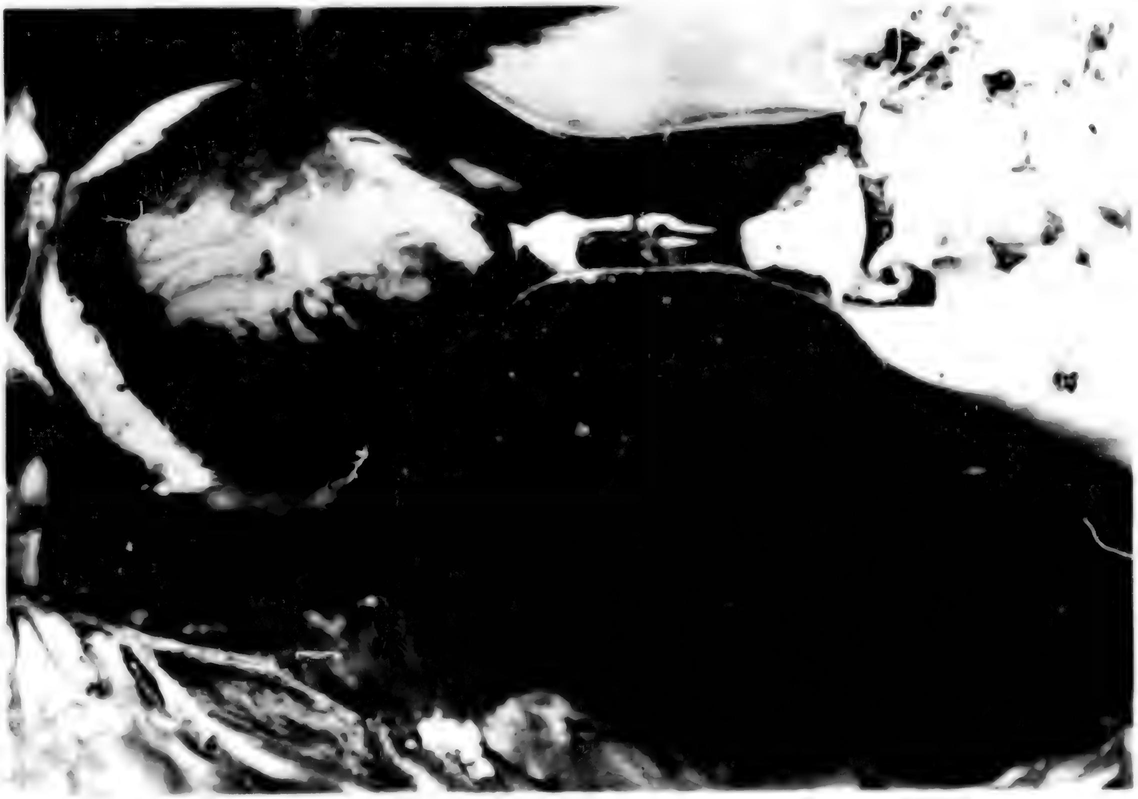
6.10b

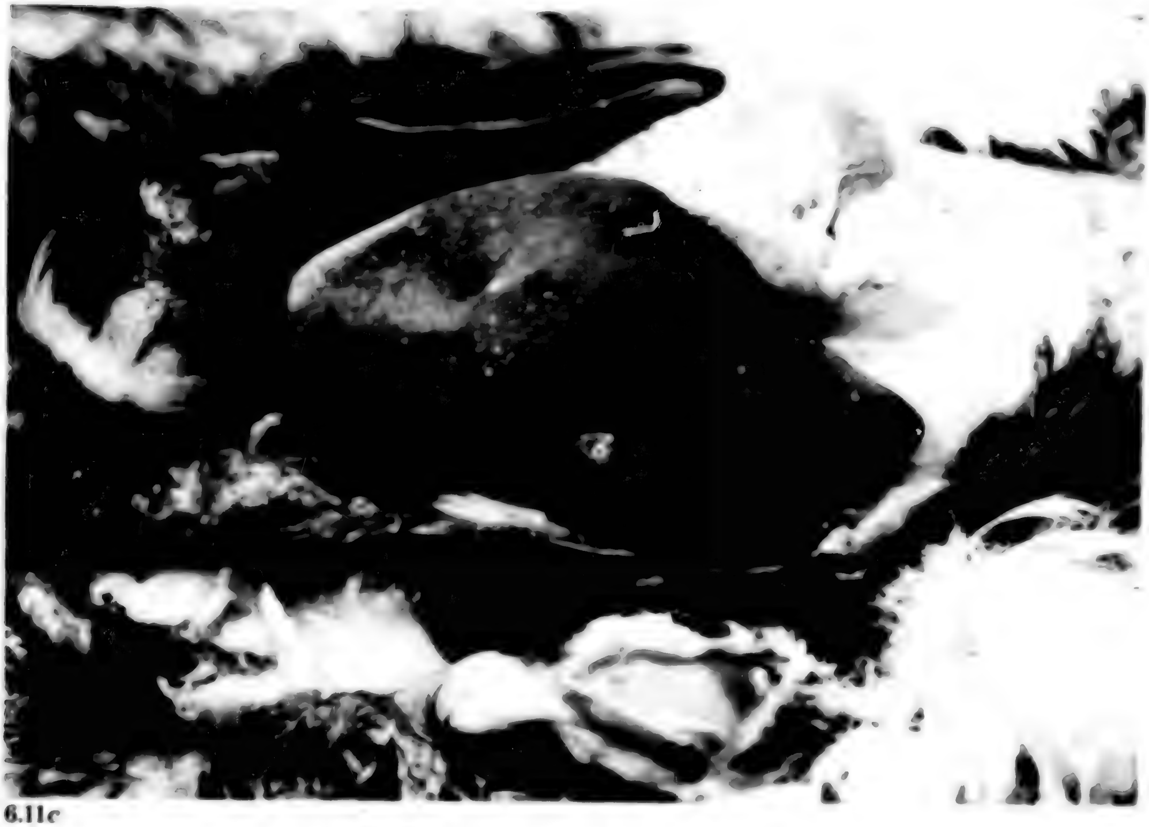
Fig. 6.10. Hemorrhages of varying degrees of severity are often seen on the heart of avian cholera-infected birds. *(a)* Pinhead-sized hemorrhages along fatty areas of the heart are readily evident in this bird. *(b)* Broad areas of hemorrhage also occur. *M.F.*



6.11*a*

6.11*b*





6.11*c*

Fig. 6.11. Lesions in the liver of avian cholera-infected birds generally appear as small, discrete yellowish spots (dead tissue). Note the variation in size and appearance of these lesions. Note also the absence of any apparent heart lesions in one bird (*a*), only a few minor hemorrhages on the coronary band of another bird (*b*), and more extensive hemorrhages on the heart muscle of the third bird (*c*). Also note the abundance of fat covering the gizzards of all these birds. The presence of this fat attests to the excellent condition these birds were in before exposure to the bacterium and to the rapidity with which each bird died. *M.F.*

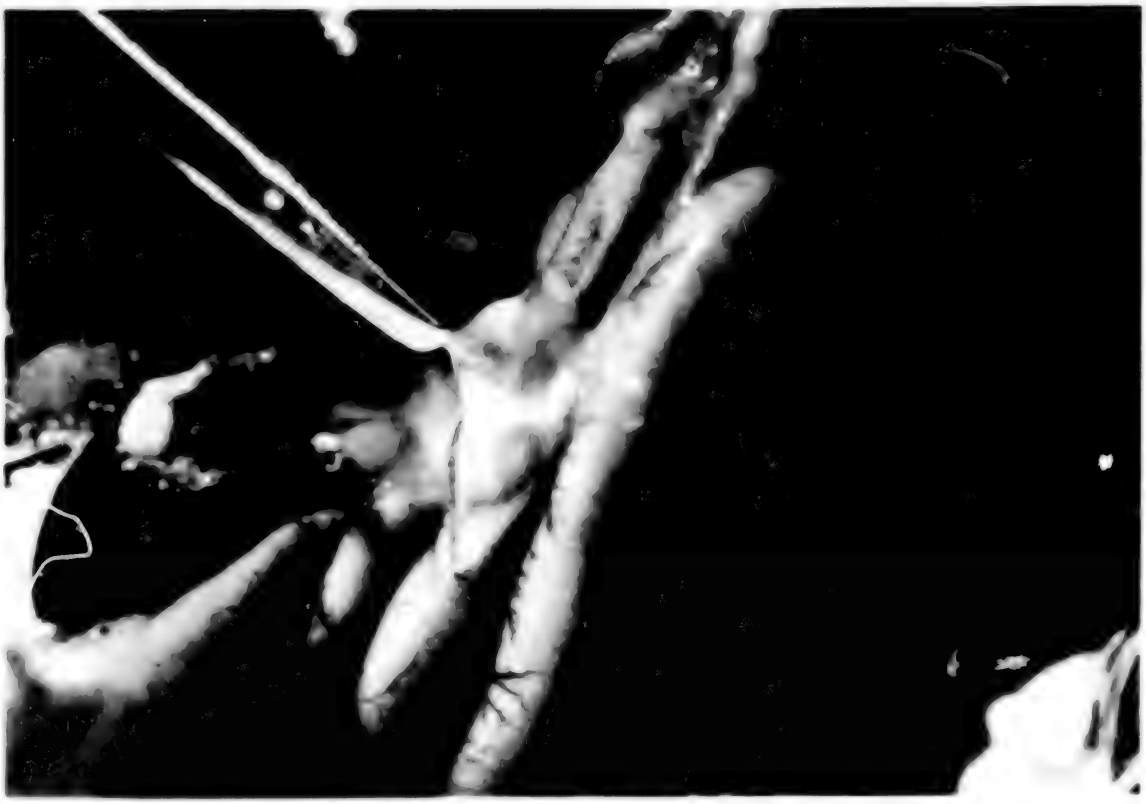


Fig. 6.12. The thickened, yellowish fluid present in the intestines of this avian cholera infected bird contains millions of bacteria. These bacteria contaminate the environment as the carcass decomposes or is scavenged, serving as a source of infection for other wildlife. *MF*

collection contributes to avian cholera control in several ways. Several milliliters of fluids containing large concentrations of *P. multocida* are often discharged from the mouths of birds dying from this disease, resulting in heavy contamination of the surrounding area. Carcass decomposition results in additional contamination. These carcasses serve to attract (decoy) other birds, thereby increasing the probability for infection. Scavenging of carcasses also results in disease transmission through the direct consumption of diseased tissue (oral exposure).

Care must be exercised during carcass collection to minimize the amount of fluid discharged into the environment from the mouths of birds. Pick birds up head first, preferably by the bill, and immediately place in plastic bags. Take care to avoid contaminating new areas while carcasses are transported to the disposal site. Double-bagging is recommended to prevent fluids leaking from punctures that may occur in the inner bag. Bags of carcasses should always be securely closed before being removed from the area.

Prompt carcass removal also prevents scavenging by birds that can mechanically transport infected material to other sites or by feeding or drinking at other locations following consumption of infected tissue. This situation is aggravated by apparent longer disease incubation times in gulls, crows, and some other avian scavengers. Instead of dying within hours or 1 to 2 days after exposure to virulent strains of *P. multocida*, death more typically occurs after several days to 1 to 2 weeks. Death may occur at locations far from the site where the bird was exposed. When these birds die, they serve as new potential focal points for contamination.

In some instances, population reduction of gulls and crows has been used to limit the role of these species in the spread and transmission of avian cholera. This technique has limited application and is not recommended as normal operating procedure. To be most effective, population reduction must be undertaken before there is a major influx of scavengers in response to carcass availability. Also, techniques used must not result in dispersal of infected birds out of the area.

Population reduction of infected American coots, gulls, terns, and eiders has also been used to combat avian cholera. Destruction of migratory birds infected with this disease can be justified only under special circumstances and conditions: (1) the outbreak must be discreet and localized rather than generalized and widespread, (2) techniques must be available that will allow complete eradication without causing widespread dispersal of potentially infected birds; (3) methods used must be specific for target species and pose no significant risk for nontarget species, (4) eradication must be justified on the basis of risk to other populations if the outbreak is allowed to continue, and (5) the outbreak represents a new geographic extension of avian cholera into an important migratory bird population.

Habitat management is another useful tool in combating avian cholera outbreaks. In some instances it may be necessary to prevent further use of a specific wetland or impoundment because it is a focal point for infection of waterfowl migrating into the area. Drainage in conjunction with creating or enhancing other habitat within the area through water diversion (from other sources) or pumping operations serves to deny bird use of the problem area and redistributes waterfowl into more desirable habitat. Ability to add a large volume of water to a problem area can also help dilute concentrations of *P. multocida* to less dangerous levels. These actions require careful evaluation of bird movement patterns and the avian cholera disease cycle. Movement of birds infected with avian cholera from one geographic location to another site is seldom desirable.

Under extreme conditions, disinfection procedures to kill *P. multocida* may be warranted in wetlands where large numbers of birds have died during a short time period. The environmental impact of such measures needs to be evaluated and appropriate approvals obtained before these actions are undertaken. A more useful approach may be to enhance the quality of the wetland in a way that reduces the survival of *P. multocida*. How this can best be accomplished is still being investigated.

Hazing with aircraft has been successfully used to move whooping cranes away from a major outbreak of avian cholera. This type of disease prevention action can also be accomplished by other methods for other species. Eagles can be attracted to other feeding sites using road-killed deer as a food source, and waterfowl can be held at sites during certain times of the year by providing refuge and food. During an avian cholera outbreak in South Dakota a large refuge area was temporarily created to hold infected snow geese in an area by closing hunting. At the same time, a much larger population of snow geese about 10 miles away was moved out of the area to prevent transmission of the disease into that population. The area closed to hunting was reopened once the desired bird movement had occurred.

Vaccination and postexposure treatment of waterfowl have both been successfully used in combating avian cholera in Canada goose propagation flocks. The National Wildlife Health Center has developed and tested a bacterin (a killed vaccine) that totally protected Canada geese from avian cholera for the entire 12 months of a laboratory study. This product has been used for several years with good results in a Canada goose propagation flock that has a great deal of contact with free-flying wild waterfowl and field outbreaks of avian cholera. Before use of the bacterin, this same flock of Canada geese suffered an outbreak of avian cholera and was successfully treated with intramuscular injections of 50 mg of oxytetracycline followed by a 30-day regimen of 500 g of tetracycline per ton of feed.

As yet, there is no practical method of immunizing large numbers of free-living migratory birds against avian cholera. However, captive propagation flocks can be protected by this method. Endangered species can be trapped and immunized if the degree of risk warrants this action. Live vaccines should not be used for migratory birds without adequate safety testing.

Human Health Considerations

Avian cholera is not considered a high risk disease for man because of differences in species susceptibility to different strains of *P. multocida*. However, *P. multocida* infections in man are not uncommon. Most of these infections result from an animal bite or scratch, primarily from dogs and cats. The use of dogs is not recommended for picking up carcasses during avian cholera outbreaks because of potential contamination of their mouths with *P. multocida* and later exposure of people as a result of licking hands or faces. Regardless, the wisdom of wearing gloves and thoroughly washing skin surfaces is obvious when handling birds that have died from avian cholera.

Infections unrelated to wounds are also common, and in the majority of human cases these involve respiratory tract exposure. This is most apt to occur in confined areas with restricted air movement where a large amount of infected material is present. Processing of carcasses associated with avian cholera die-offs should be done outdoors or in other areas with adequate ventilation. When disposing of carcasses by open burning, avoid direct exposure to smoke from the fire.

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Chapter 7.
Avian Botulism

Louis N. Locke and Milton Friend

Synonyms

Limberneck, western duck sickness,
duck disease, alkali poisoning

Cause

Avian botulism is a paralytic, often fatal disease of birds resulting from ingestion of toxin produced by the bacterium, *Clostridium botulinum*. Seven distinct types of toxin designated by the letters A through G have been identified, along with several subtypes. Waterfowl die-offs from botulism are usually caused by type C toxin; sporadic die-offs among fish-eating birds, such as common loons and gulls, have been caused by type E toxin. Type A toxin also affects birds, usually as the cause of botulism in domestic chickens. Types B, D, F, and G are not known causes of avian botulism in North America.

Not enough is known about avian botulism to precisely identify the factors leading to an outbreak. When an outbreak does occur, it is usually perpetuated by a well-understood bird–maggot cycle (Fig. 7.1). Bacterial growth and various environmental conditions also combine to favor toxin production in wetlands.

Clostridium botulinum persists in wetlands in a spore form that is resistant to heat and drying and remains viable for many seasons and, in some instance, for years. Toxin production occurs during multiplication of the vegetative form of the bacteria following spore germination. The vegetative form requires dead organic matter and a complete absence of oxygen to grow and produce toxin. Optimum growth of the bacteria occurs at about 25 °C (77 °F). Toxin production is optimized within a pH range of 5.7 to 6.2 and depends on the protein content of the medium in which the bacteria are growing. All kinds of animal protein are suitable for toxin production. Especially potent toxin is produced in bird, mammal, and a variety of invertebrate carcasses. This entire process is further complicated by a poorly understood but important role of bacteriophages—viruses that infect bacteria. Recent findings show that bacteriophages determine if toxin will be produced during *C. botulinum* growth and multiplication stages.

Important environmental factors that contribute to initiation of avian botulism outbreaks include water depth, water level fluctuations, and water quality; the presence of vertebrate and invertebrate carcasses; rotting vegetation; and high ambient temperatures.

Shallow water permits rapid warming of the submerged marsh soil during periods of high ambient temperatures. Toxin is produced when these soils contain both the spores of *C. botulinum* and suitable organic nutrients for spore germination and reproduction of bacterial cells. Fluctuating water levels that produce "feather edge" shorelines contribute to avian botulism outbreaks when terrestrial and aquatic invertebrates die as land areas are flooded and the underwater areas subsequently become dry when the water recedes. Fertilization of a marsh with sewage or run-off from agricultural activities can stimulate plant or invertebrate animal population growth for short periods, but results in plant and invertebrate die-offs once this stimulus subsides. The resulting mass of nutrients is then available for growth of *C. botulinum* and toxin production. Dense vegetation can entrap and thus kill fish, amphibians, or invertebrates, and masses of rotting marsh plants can reduce oxygen levels to the point that aquatic animal life is killed. Both of these conditions provide large amounts of growth material for toxin production. The presence of vertebrate carcasses and high ambient temperatures are also conducive to the buildup of fly populations involved in the bird–maggot cycle for avian botulism transmission.

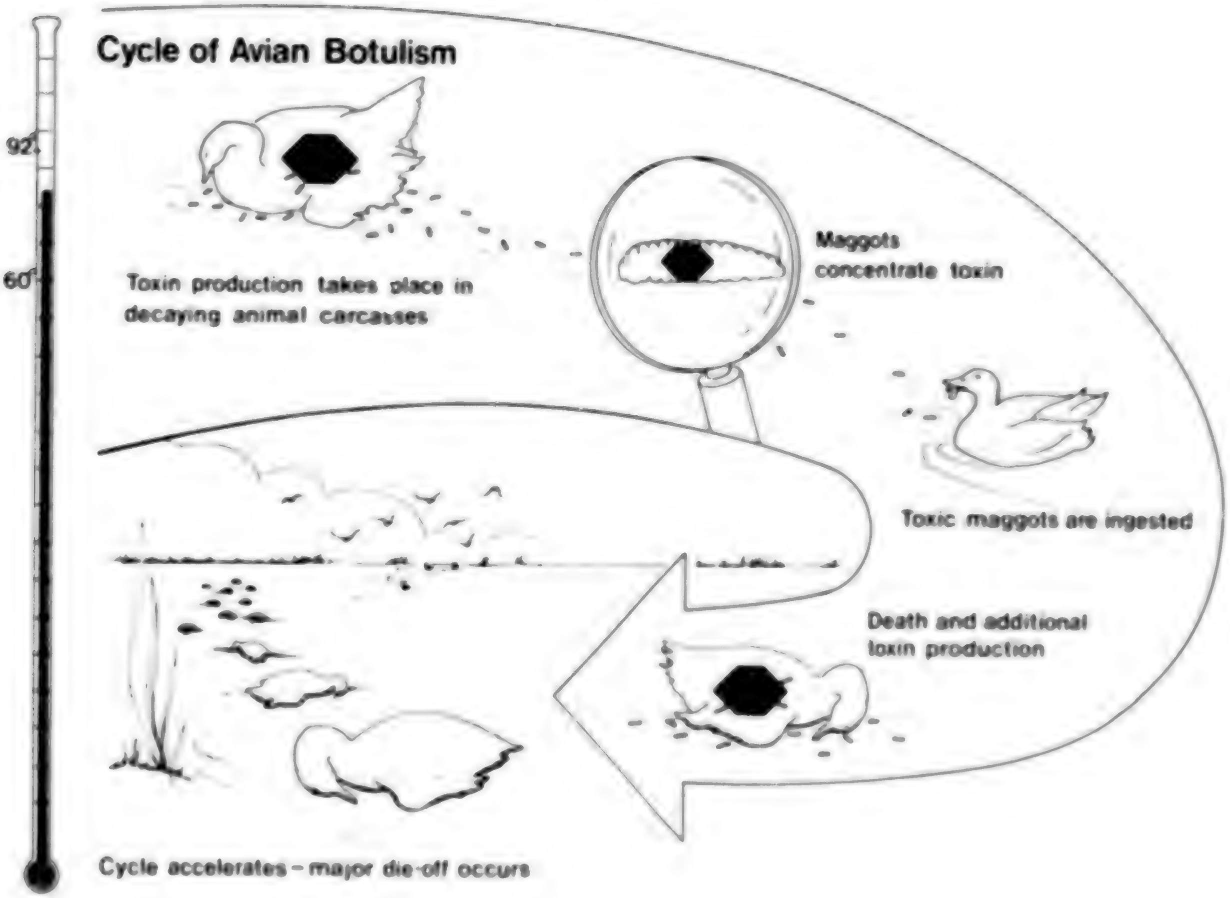

Cycle of Avian Botulism
92°
60°
Toxin production takes place in decaying animal carcasses
Maggots concentrate toxin
Toxic maggots are ingested
Death and additional toxin production
Cycle accelerates – major die-off occurs

Fig. 7.1. Avian botulism cycle

Species Affected

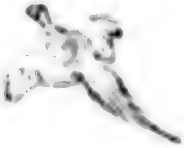
Many species of birds and some mammals are affected by type C botulism. In the wild, waterfowl and shorebirds are most often affected (Fig. 7.2); in captivity, waterfowl, pheasants, and ranch mink are most frequently affected (Fig. 7.3). Vultures are known to be highly resistant to type C toxin. Although dogs and cats are usually regarded as being resistant to type C toxin, a few cases have been reported in dogs. Recently, type C botulism occurred in captive African lions that were fed chickens that had died of type C botulism.

Losses vary a great deal from year to year at site-specific locations and from species to species. A few hundred birds may die in 1 year and tens of thousands or more the following year. More than a million deaths from avian botulism have been reported in relatively localized outbreaks in a single year, and outbreaks with losses of 50,000 birds or more are relatively common (Table 7.1). Avian botulism is one of the three most important disease problems of migratory birds, perhaps the most important.

Distribution

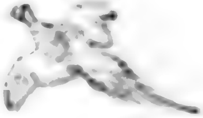
Outbreaks of avian botulism have occurred in the United States and Canada since the beginning of the century, if not earlier. Outbreaks have also been reported to occur in many other countries. Most of these reports are recent, usually within the past 20 years (Table 7.2). Most type C outbreaks within the United States occur west of the Mississippi River; however, outbreaks have occurred from coast to coast and border to border (Fig. 7.4). Type E outbreaks in birds are much less frequent and within the conterminous United States have been confined to the Great Lakes region.

Fig. 7.2 Frequency of botulism in major groups of wild birds.

Type of bird	Type C	Type E
Waterfowl	● ● ● ●	●
Loons	○	● ● ●
Herons	● ●	○
Shorebirds	● ● ● ●	○
Gulls	● ● ●	● ● ●
Raptors	●	○
Gallinaceous 	●	○
Songbirds	●	○

● ● ● ● Common; die-offs occur almost yearly
● ● ● Frequent
● ● Occasional
● Infrequent
○ Not reported

Fig. 7.3. Frequency of botulism in captive birds.

Type of bird	N. America	Europe
Waterfowl	●	●
Pheasants 	● ● ●	● ●
Domestic chickens	○	● ● ●
Songbirds	○	● ●

● ● ● Frequent
● ● Occasional
● Infrequent
○ Rare or not reported

Seasonality

July through September are the primary months for type C avian botulism outbreaks in the United States and Canada. However, outbreaks occur as late as December and January and occasionally during early spring in southern portions of the United States and in California. Type E outbreaks have occurred during late fall and spring.

Field Signs

Lines of carcasses coinciding with receding water levels generally typify the appearance of major die-offs, although outbreaks have also occurred in impoundments containing several feet of water, lakes with stable water levels, and in large rivers. When receding water conditions are involved, botulism is typically a disease of the water's edge, and seldom are sick or dead birds found very far from the vegetation bordering the water or the original water's edge (Fig. 7.5). In impoundments where water levels are relatively stable, affected birds are likely to be found in areas of flooded vegetation. Botulism-affected birds also tend to congregate along vegetated peninsulas and islands (Fig. 7.6).

Healthy birds, sick birds, and recently dead birds will commonly be found together during a botulism outbreak, along with carcasses in various stages of postmortem decay. Often, a variety of species representing two or three or even more orders of birds suffer losses simultaneously.

Avian botulism affects the peripheral nerves and results in paralysis of voluntary muscles. Inability to sustain flight is seen early in botulism, but this sign is not useful in separating botulism from other diseases. Once the power of flight is lost and

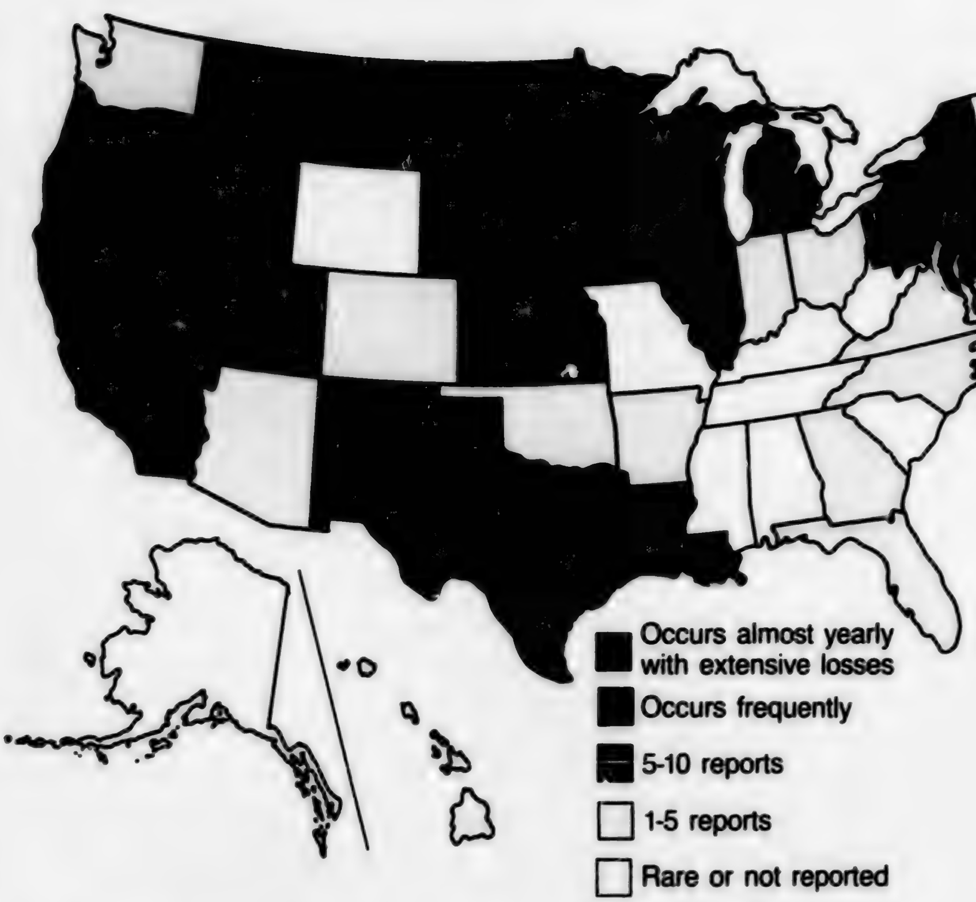


Fig. 7.4. Frequency of type C botulism in waterfowl.

paralysis of leg muscles has occurred, ducks suffering from botulism often propel themselves across the water and mud flats with their wings (Fig. 7.7; see also Figure 1.3 in chapter on Recording and Submitting Specimen Data). This sequence of signs contrasts with that of lead-poisoned birds, which retain their ability to walk and run even though flight becomes difficult.

Paralysis of the inner eyelid or nictitating membrane (Fig. 7.8) and neck muscles follows, resulting in inability to hold the head erect (Fig. 7.9). These are the two most easily recognizable signs of avian botulism. Once birds reach this stage, death from drowning often occurs before the bird might otherwise die from the respiratory failure caused by botulinum toxin.

Avian botulism often occurs in the seasons when waterfowl are flightless because of wing molt. Care then must be taken to separate birds in molt from those with early stages of intoxication because the behavior of these birds may be similar. Molting birds are difficult to catch and birds that cannot be captured with a reasonable effort should not be pursued further. If these birds are suffering from botulism, they can be easily captured when they become unable to dive to escape pursuit. Birds at this level of intoxication still have a high probability for survival if proper treatment is administered.

Gross Lesions

There are no characteristic or diagnostic gross lesions in waterfowl dying of type C or type E botulism. Often, affected birds die by drowning, and lesions associated with drowning may be present.

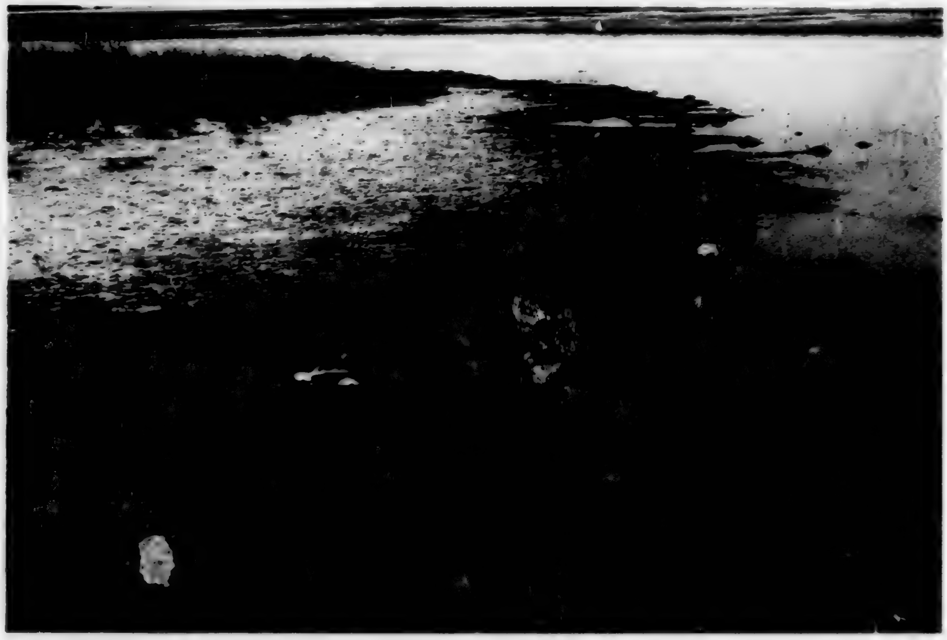


Fig. 7.5. Typical scene of avian botulism. Dead birds are often found along the shore in parallel rows that represent receding water levels. *J.R.*

Fig. 7.6. Botulism-affected birds tend to congregate along vegetated peninsulas and islands. Both dead and sick birds are evident in this photograph. *M.F.*

Table 7.1. *Major waterfowl botulism outbreaks.*

Location	Year	Estimated loss
Utah and California	1910	millions
Lake Malheur, Oregon	1925	100,000
Great Salt Lake, Utah	1929	100,000–300,000
Tulare Basin, California	1941	250,000
Western United States	1952	4–5 million
Montana (near Billings)	1978	50,000
Montana (near Billings)	1979	100,000
Great Salt Lake, Utah	1980	110,000

Table 7.2. *Initial outbreaks by location of type C avian botulism in wild waterfowl.*

Location	Year
The Americas	
United States	1910
Canada	1913
Uruguay	1921
Mexico	1976
Australia–Asia	
Australia	1934
New Zealand	1972
Japan	1973
Europe–Africa	
Denmark	1967
Great Britain	1969
Netherlands	1970
East Germany	1971
West Germany	1971
Italy	1973
Spain	1973
Union of South Africa	1956

Diagnosis

The most reliable test for avian botulism is the mouse protection test. Blood is collected from a sick or freshly dead bird and the serum fraction is then inoculated into two groups of laboratory mice, one group of which has been given type-specific antitoxin. The mice receiving antitoxin will survive and those that receive no antitoxin will become sick or die with characteristic signs if botulinum toxin is present in the serum sample.

A presumptive diagnosis is often based on a combination of signs in sick birds and the absence of obvious lesions of disease when the internal organs and tissues of sick and dead birds are examined. However, this initial diagnosis must be confirmed by the mouse protection test to separate avian botulism from algal poisoning, castor-bean poisoning, and other toxic processes.

Control

Management of Environment. Control efforts need to focus on three important factors that contribute to the development and maintenance of avian botulism outbreaks: fluctuating water levels during hot summer months, an abundance of flies, and animal carcasses for toxin production. On areas to be managed primarily for migratory waterfowl (ducks, geese, swans), reflooding of land that has been dry for a long time is not recommended during the summer months. Similarly, sharp drawdowns of water should be avoided since they could result in fish-kills and die-offs of aquatic invertebrates whose carcasses could then become centers for the growth of *C. botulinum*. On those areas managed primarily for shorebirds, water drawdowns are essential, and botulism control must focus on a cleanup of any carcasses that may result.

Prompt removal and proper disposal of vertebrate carcasses by burial or burning are highly effective mechanisms for removing the major sources of toxin production and maggot development. The importance of prompt and thorough carcass removal and proper disposal cannot be overemphasized. Several thousand toxic maggots can be produced from a single waterfowl carcass (Fig. 7.10). Consumption of as few as two to four of these toxic maggots can result in intoxication of a duck, thereby perpetuating the botulism cycle. It is not uncommon to find three or four freshly dead birds within a few feet of a maggot-laden carcass. Failure to carry out adequate carcass removal and disposal programs can result in a rapid buildup of highly toxic materials, and can accelerate losses as well as seed the environment with *C. botulinum* toxin and spores as the carcasses decompose. Toxin formed in these carcasses is quite stable. This preformed toxin can be taken in by invertebrates, remain free in bottom sediments, or become

Fig. 7.7. Botulinum-intoxicated birds that have lost the power of flight and use of their legs often attempt escape by propelling themselves across water or land using their wings. *M.F.*

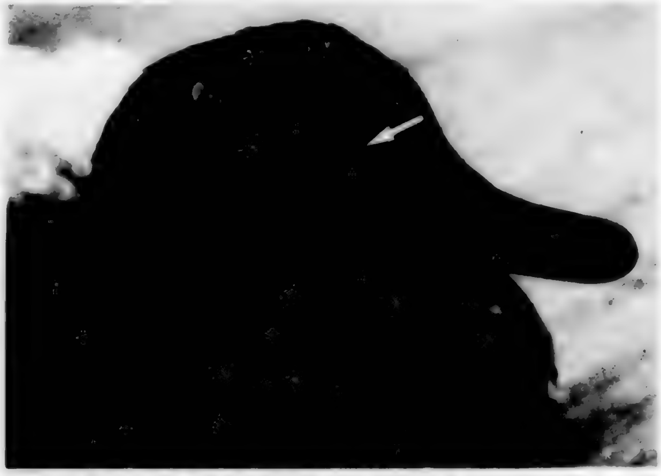


Fig. 7.8. Paralysis of the inner eyelid is a common sign in botulinum-intoxicated birds. *J.R.*

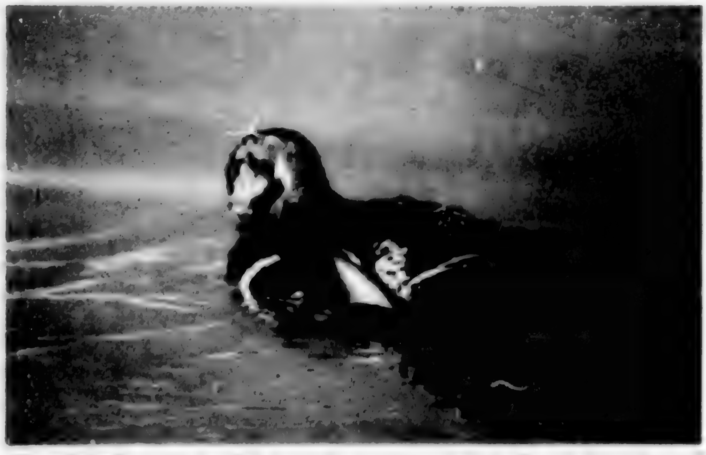
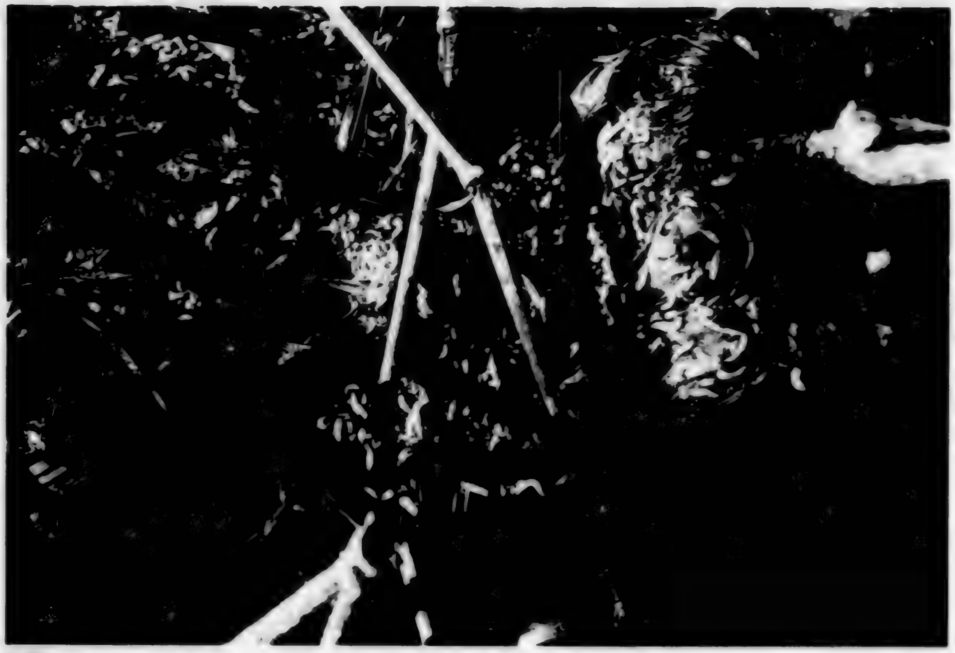


Fig. 7.9. Paralysis of the neck muscles in botulinum-intoxicated birds results in inability to hold the head erect (limberneck). Death by drowning often results. *M.F.*



7.10*a*

7.10*b*

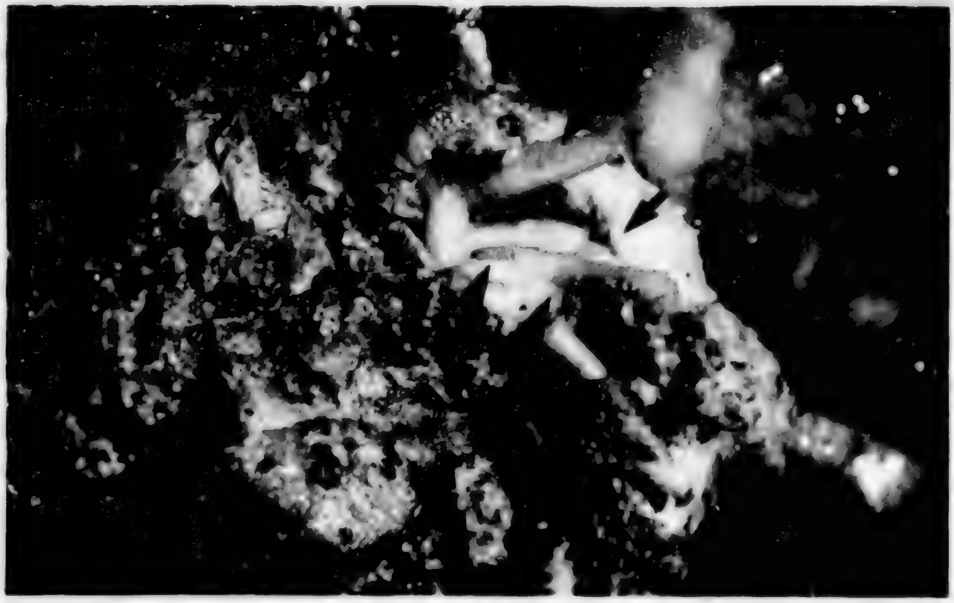


Fig. 7.10. (*a*) Several thousand toxic maggots can be produced from a single waterfowl carcass. (*b*) Waterfowl and other birds readily feed on these maggots as exemplified by their presence in the gizzard of this duck that died from avian botulism. *M.F.*

suspended in the water column where it can serve as the source of winter and spring botulism outbreaks when ingested by feeding birds.

Many botulism outbreaks occur on the same wetlands year after year, and within a wetland there may be localized "hot spots." Also, outbreaks often follow a fairly consistent and predictable time frame. These conditions have direct management implications that should be applied toward minimizing losses. Specific actions that should be taken include accurately documenting conditions and dates of outbreaks in problem areas, planning for and implementing intensified surveillance and carcass pickup and disposal, and modifying habitats to reduce the potential for botulism losses and deny bird use on major problem areas during the botulism "season." Surveillance and carcass disposal activities should start 10 to 15 days before the earliest documented cases and continue 10 to 15 days after the end of the botulism "season." Habitat modifications will primarily involve control of water quality and water levels.

Because fish carcasses can also serve as sites for *C. botulinum* growth, they should be promptly removed during fish control programs in marsh environments or fish control programs should be restricted to the cooler months of the year, (non-fly season). Power lines that cross marsh environments have been associated with major botulism outbreaks. Bird carcasses from collisions with power lines have served as initial points for toxin production within the marsh environment. Therefore, if possible, power lines should not be placed across marsh environments used by large concentrations of water birds.

Numerous outbreaks of avian botulism have been associated with sewage and other wastewater discharge into marsh environments. This relation is not presently understood, but has occurred often enough that wetland managers should discourage the discharges of these effluents when substantial waterfowl or shorebird use is occurring or is likely to occur on an area during the ensuing 30 days.

Treatment of Sick Birds. Studies at Bear River Refuge, Utah, have clearly demonstrated that a high percentage of botulinum-intoxicated waterfowl can be saved. If the birds are provided with fresh water and shade, or injected with antitoxin, recovery rates of 75–90% and higher can result (Fig. 7.11). In contrast with waterfowl, very few American coots, shorebirds, gulls, and grebes have survived treatment for botulism. Experience to date with these species indicates that rehabilitation efforts are not worthwhile.

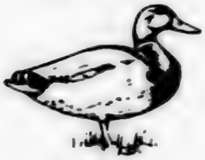
When botulinum-intoxicated birds are treated, the birds should be maintained under conditions that provide unrestricted access to fresh water, maximum provision for shade, an opportunity for birds that recover to fly out of the enclosure when they choose to, and minimum disturbance (including presence of humans). It is also important to remove carcasses daily from enclosures to prevent the buildup of toxic maggots within the treatment area, and to monitor the cause of mortality since one cannot assume botulism is the cause. The weakened condition of botulinum-intoxicated birds can result in the eruption of infectious disease such as avian cholera. Should this occur, it is essential to immediately address the infectious disease problem.

Costs associated with capturing and treating sick birds are high. Therefore, the emphasis for dealing with avian botulism should be on prevention and control of this disease rather than on treatment of intoxicated birds. However, antitoxin should be available for use in case endangered species are affected.

Human Health Considerations

Botulism in humans is usually the result of eating improperly home-canned foods and is most often caused by type A or type B botulinum toxin. There have been a few human cases of type E botulism in North America as the result of eating improperly smoked or cooked fish or marine products. Although humans are regarded as being fairly resistant to type C botulinum toxin, at least two cases of type C botulism have been reported in man, although the origins were unidentified. Thorough cooking destroys botulinum toxin in food.

Fig. 7.11. Recovery rates of ducks in response to antitoxin therapy

Duck type	Amount of antitoxin (cc)	Clinical signs (% recovery)	
		Moderate	Severe
Pintail	0.5	91.9	69.0
	1.0	93.5	73.3
G-W Teal	0.5	93.5	57.4
	1.0	91.2	58.1
 Mallard	0.5	81.6	66.3
	1.0	84.9	67.8
Shoveler	0.5	89.2	53.1
	1.0	95.1	57.6

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Chapter 8
Avian Tuberculosis

Thomas J. Roffe

Synonyms

Mycobacterium avium, mycobacteriosis, tuberculosis, TB

Cause

Avian tuberculosis is usually caused by the bacterium *Myobacterium avium*. At least 20 different types of *M. avium* have been identified, only three of which are known to cause disease in birds. Other types of *Mycobacterium* rarely cause tuberculosis in most avian species. However, parrots, macaws, and other large psittacines are susceptible to human and bovine types of tuberculosis bacilli and ornamental game birds are very susceptible to bovine types of the bacteria. Transmission generally occurs by direct contact with infected birds, ingestion of contaminated feed and water, or contact with a contaminated environment.

Species Affected

All avian species are susceptible to *M. avium*. Man, most species of livestock, and a variety of other mammals can also become infected. The dog appears to be quite resistant to the avian type of tuberculosis (Fig. 8.1). In captivity, turkeys, pheasants, quail, cranes, and certain birds of prey are more commonly affected than waterfowl. However, once established, tuberculosis can be a common and important disease in captive waterfowl flocks. In free-living wild birds, the highest prevalence is among species that live in close association with domestic stock (e.g., house sparrows and European starlings) and in scavengers (e.g., gulls). The prevalence of tuberculosis in free-living waterfowl has not been determined, although generally less than 1% of birds examined at postmortem are affected. Advanced disease and clinical signs are seen most often in adult birds because of the chronic, insidious nature of avian tuberculosis.

Distribution

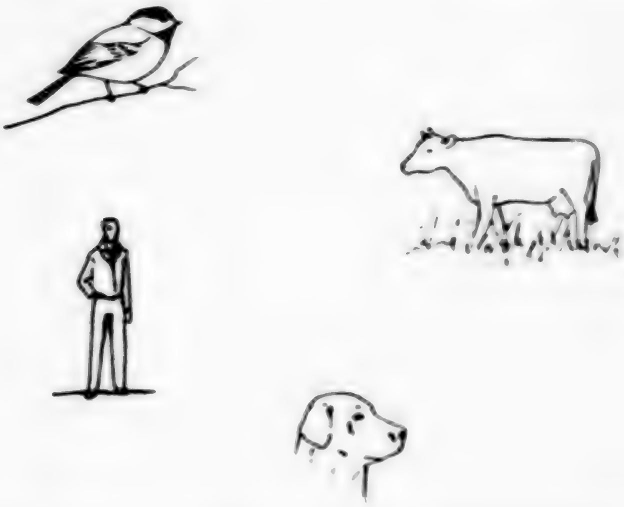
Avian tuberculosis is a ubiquitous and cosmopolitan disease of free-living, captive, and domestic birds. The disease occurs most commonly in the North Temperate Zone. Distribution of this disease in free-living migratory birds is inferred from birds submitted for necropsy and underrepresents both the geographic distribution and frequency of infection for individual species. Avian tuberculosis likely exists in small numbers of free-living migratory birds wherever there are major migratory concentrations.

Seasonality

Seasonal trends of tuberculosis in wild birds have not been documented. The chronic nature of this disease guarantees its presence at all times of the year for both wild and captive birds.

Factors that may influence seasonal occurrences of tuberculosis in migratory birds are changes in habitat used, food base during the year, and interspecies contacts. Predatory and scavenger species such as raptors and crows often have different opportunities for food items during different periods of the year, thus possibly influencing exposure. Also, contaminated sewage and wastewater

Fig. 8.1. Relative susceptibility of various animal groups to *M. avium*.



Species group	Relative susceptibility
Birds	● ● ●
Livestock	● ●
Man	●
Dogs	○

● ● ● Highly susceptible
● ● Moderately susceptible
● Slightly susceptible
○ Highly resistant

environments containing tubercle bacilli are more likely to be used by waterfowl during fall and winter than during warmer months. Wastewater sites are often closed to hunting, thereby serving as refuge areas, or discharge warm water that maintains open water in subfreezing temperatures, thus inviting ready use by waterfowl. Contact between migratory birds and poultry and livestock is often restricted to specific periods of the year due to husbandry practices.

Environmental conditions can greatly affect the prevalence of tuberculosis in captive birds. Captive birds on an inadequate diet maintained in crowded, wet, cold, poorly ventilated, and unhygienic aviaries have increased susceptibility to tuberculosis.

Field Signs

No clinical signs specifically identify avian tuberculosis in birds. Infected birds are often emaciated, weak and lethargic, and exhibit wasting of the muscles. These signs are similar to those of lead poisoning and other debilitating conditions. Other signs depend on which body system is affected and include diarrhea, lameness, and unthrifty appearance. Darkening and dulling of plumage have been reported in the United Kingdom for wood pigeons infected with tuberculosis, but not for waterfowl or other species. Abscesses and nodular growths have been reported on the skin of birds in the same locations where pox lesions are commonly seen—around the eyes, at the wing joints, on the legs, side of the face, and base of the beak. Other birds have died of avian tuberculosis without any obvious clinical signs or external lesions.

Gross Lesions

Typical cases of avian tuberculosis in migratory birds involve emaciated carcasses having solid to soft or crumbly, yellow to white or grey nodules (< 1 mm to several centimeters) embedded deep in infected organs and tissues. The liver (Fig. 8.2*a*) is most often involved in waterfowl, but spleen (Fig. 8.2*b*), lung, and intestines (Fig. 8.2*c*) may also contain similar nodules. Aggregations of these nodules may appear as firm, fleshy, grapelike clusters.

Nodular lesions of tuberculosis are often grossly similar to those of aspergillosis and require a laboratory diagnosis to differentiate the two diseases, as well as other diseases that produce similar lesions. Less typical lesions can also exhibit the appearance of other diseases. Sometimes the primary lesions seen at necropsy are enlarged livers and spleens that are so fragile they easily rupture upon being handled. Most of these cases have livers and spleens with a tan to green translucence due to amyloid deposits. Less commonly, in situations where nodules are not formed nor is amyloid deposited, the liver and spleen can be large, pale, and firm.

Diagnosis

Typically, tuberculosis is discovered in captive birds during routine investigation of mortality, and in wild birds during carcass examinations associated with die-offs due to other causes. The gross lesions described above (Fig. 8.2) are suggestive of tuberculosis, but a definitive diagnosis is based on bacteriological isolation and identification of the organism. Since *M. avium* is slow-growing and other bacteria can easily overgrow it, a noncontaminated sample is needed for examination. Whole carcasses are preferred, but when this is not possible, remove the leg at the hip joint for submission. The leg should be wrapped in clean aluminum foil and placed in a plastic bag and frozen for shipment to a qualified disease diagnostic laboratory. The marrow within the femur has the lowest potential for being contaminated, while providing a good sample for the bacteriologist.

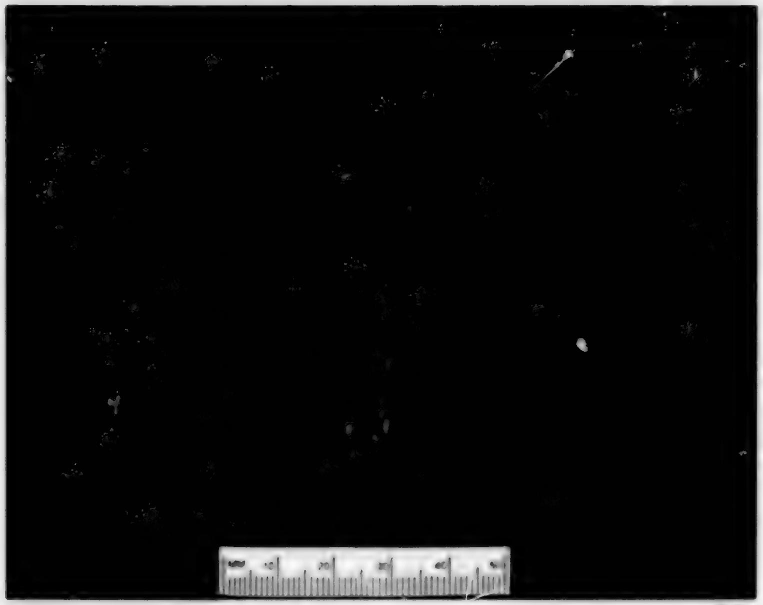
Isolation of the bacteria can also be made from infected tissues (those with gross lesions) and microscopic studies can provide a diagnosis of tuberculosis, although the species of *Mycobacterium* cannot be determined in the latter instance. Because this disease is transmissible to man, extra care must be taken when handling infected carcasses. In situations where carcass or tissue submissions to a laboratory are not possible within a short time, tissue preserved in 10% buffered formalin solution is useful for diagnostic purposes (see chapter on Specimen Collection and Preservation).

Control

Tuberculosis is difficult to detect in free-living migratory birds despite the broad geographic distribution of this disease. Mortality rarely occurs as a major die-off and there are no practical testing procedures for free-living wild birds. Therefore, there is no focal point and, hence, no method developed for disease control in wild bird populations. By contrast, tuberculosis can be a serious problem in captive migratory birds and has been reported as such in sea ducks. Some captive flocks of ducks and geese have experienced losses of nearly 30% from tuberculosis.

Close monitoring of the health of migratory bird populations—free-living or captive—is an essential first step in detecting tuberculosis so that control efforts can be developed and initiated when feasible. This can best be accomplished through the timely submission of carcasses to disease diagnostic laboratories. Tuberculosis testing of birds maintained in captivity, and laboratory analyses of fecal samples from captive and wild flocks also can be used to identify the presence of this disease. These tests do not detect all infected birds, but are useful in identifying infected flocks.

Fecal contamination of the environment is the major means of tuberculosis dissemination; ingestion of the bacterium in contaminated feed and water is the most common means of disease transmission. Because this bacterium can survive in an organic substrate outside of the vertebrate host for a year or more, a few infected individuals



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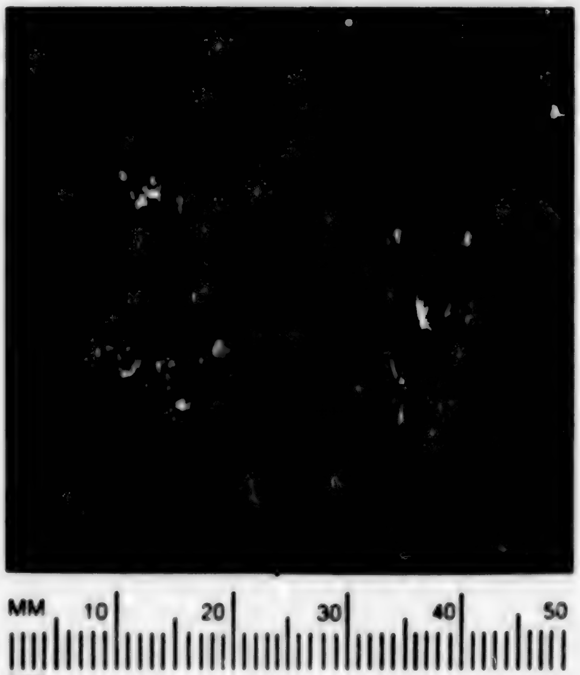
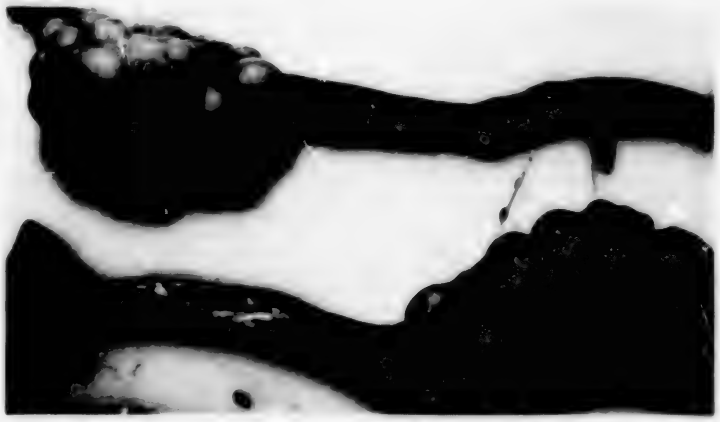


8.2b

Fig. 8.2. The raised, firm nodules in these organs are typical lesions of avian tuberculosis; (*a*) liver; (*b*) spleen; and (*c*) intestine. *J.R.*

8.2c



can contaminate an area that has prolonged bird-use patterns. Site contamination can also occur as a result of wastewater discharges containing the bacterium. Tuberculosis outbreaks have been associated with sewage effluents and discharges from slaughter houses, meat processing plants, and dairies. In one instance, an outbreak occurred in a captive waterfowl flock when contaminated water was sprayed into the enclosure. These events illustrate the importance of prevention to control tuberculosis in free-living and captive migratory birds.

The use of wastewater for maintaining captive waterfowl and other migratory birds is questionable without adequate testing and treatment to assure that the water does not contain tubercle bacilli. Similar considerations should be given to the use of wetlands for wastewater discharges and the use of wastewater to create wetlands for migratory bird habitat.

Infected flocks of captive birds should be destroyed because treatment is ineffective and not all infected birds will be detected by current testing procedures. Because of the long-term environmental persistence of the tubercle bacilli, additional bird use of the site should be avoided for about 2 years. Vegetation removal and turning of the soil several times during this period will facilitate sunlight-induced environmental decay of the bacilli. Eradication of free-living migratory flocks is rarely feasible. However, in instances where a major outbreak of tuberculosis occurs in wild birds, an assessment should be made of the circumstances, and limited population reduction should be considered if the remaining population at-risk is well defined, limited in immediate distribution, and involves species that can sustain this action. Habitat manipulation (such as drainage) and scaring devices (such as propane exploders) can be used in some instances to deny birds use of areas where tuberculosis outbreaks occur.

Human Health Considerations

There are many authenticated cases of *M. avium* infection in people, although humans are considered highly resistant to this organism. The disease is generally considered noncontagious from infected individuals. Infection is more likely to occur in persons with preexistent diseases, especially those involving the lungs, and in persons whose immune systems are impaired by illness or therapy (e.g., steroids).

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Chapter 9.
Avian Salmonellosis

Richard K. Stroud and Milton Friend

Synonyms

Salmonellosis, pullorum disease, fowl typhoid, paratyphoid[1]

Cause

Avian salmonellosis is a group of diseases rather than a specific disease. All are caused by bacteria belonging to the genus *Salmonella*. About 1,800 to 2,000 serotypes exist based on various combinations of antigens present as part of the bacterial cell. These bacteria are also differentiated on the basis of their motility. Pullorum disease and fowl typhoid are both distinctive diseases caused by naturally nonmotile forms of salmonellae. Both diseases are relatively host specific and neither is an important cause of illness or death for free-living species of wild birds.

Motile forms of salmonellae that infect birds are generally referred to as "paratyphoid organisms" and are frequently the cause of illness and death in wild birds. About 200 paratyphoid serotypes have been isolated just from chickens and turkeys in the United States. Paratyphoid organisms can infect all other warm- and cold-blooded vertebrates, including man (Table 9.1).

This chapter focuses on "paratyphoid infections" in wild birds. These infections can be transmitted in many ways, including fecal contamination of eggs, food, and water by carrier birds; transmission by rats, mice, and other mammals; inhalation during hatching and brooder operations; exposure to contaminated environments; ingestion of poultry and bird feeds containing contaminated animal byproducts; and direct contact with infected birds and animals.

Species Affected

Paratyphoid infections occur in most species of warm- and cold-blooded vertebrates, are more prevalent in turkeys than in any other domestic birds, and are common in young domestic geese and ducks, pigeons, and many species of wild birds (Fig. 9.1).

Salmonella typhimurium is the most common paratyphoid infection found in birds. Isolations are often made from apparently healthy birds and from birds with other disease conditions. Illness or death in wild birds from paratyphoid infections most frequently involves small numbers of cases. However, mortality of die-off proportions has also been reported. These events are usually associated with passerine birds such as house sparrows, evening grosbeaks, Northern cardinals, pine siskins, and American goldfinches at backyard bird feeding stations. Losses of European starlings, brown-headed cowbirds, blackbirds, common grackles, and mourning doves have also been associated with *S. typhimurium* infections at bird feeders. Die-offs of aquatic birds due to salmonellosis have involved mute swans, ducks, American coots, and gulls.

Distribution

Paratyphoid infections have been found in wild birds from New Zealand, Australia, Antarctica, Europe, Canada, and other nations in addition to the United States. Infections of poultry exist wherever poultry are raised, thereby attesting to the worldwide distribution of these bacteria in avian species. Within the United States, infections in wild birds have been most frequently recognized in midwestern and eastern regions. In Europe, most reports of paratyphoid infections in migratory birds are associated with sewage-contaminated habitat near urban areas.

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[1] Although all these names are synonyms for salmonellosis, each is a distinct clinical variant of salmonellosis caused by a different type of salmonella.

Table 9.1. *Three of the most common Salmonella spp. affecting birds.*

Common name	Causative agent	Principal species affected	Primary age groups affected
Pullorum disease	*Salmonella pullorum*	Domestic chickens and turkeys	2-3-week-old chicks
Fowl typhoid	*Salmonella gallinarum*	Domestic chickens and turkeys	Growing and adult birds
Paratyphoid infection	*Salmonella typhimurium*	Domestic turkeys; wild gulls and songbirds	All ages

Seasonality

There is no known seasonal pattern to salmonellosis outbreaks. Instead, they are associated with environmental conditions that facilitate disease transmission, and with reduced host resistance. For example, winter outbreaks at backyard feeders probably result from contamination of feeding platforms and feed by fecal deposits from *Salmonella* carriers in the population. Sewage contamination of wetlands may result in local outbreaks at any time of year.

Field Signs

There are no distinctive signs associated with paratyphoid infections in wild birds. Different species and ages of birds may have different signs even if infected with the same serotype of *Salmonella*. Dead or dying birds at bird feeders are often thought to be "poisoned" because of rapid death. Weakness, drowsiness, depression, and diarrhea have been observed in poultry and captive flocks. Young domestic ducklings usually die slowly. They tremble and gasp for air, their wings often droop and they sometimes stagger and fall over just before death. These birds often have pasted vents and eyelids that are swollen and stuck together by a fluid discharge. Convulsions and other neurological signs often precede death in songbirds observed dying in zoos and at backyard feeders.

Adult poultry rarely exhibit clinical signs of paratyphoid infection; a general unthrifty appearance and watery diarrhea are the most probable signs of disease. In contrast, the occurrence of illness and death from paratyphoid infections in adult wild birds is relatively common.

Gross Lesions

The occurrence and types of gross lesions are highly variable depending on the course of infection, the virulence of the organism, and the host's resistance. In acute cases, obvious lesions can be completely absent. Livers often become swollen and crumbly, showing small reddened or pale spots if the course of the disease has been prolonged. In other infections, so-called paratyphoid nodules develop in the liver and extend into the body cavity. These are small, tan-to-white, granular nodules that are best seen under a microscope. An acute intestinal infection can be recognized by reddened internal lining of the posterior two-thirds to one-half

Fig. 9.1. Relative occurrence of three types of salmonellosis in selected avian species.

Species affected	Pullorum disease	Fowl typhoid	Paratyphoid infection
Ducks/geese/swans	● ●	a	● ● ●
Cranes	○	○	● ●
Gulls	○	○	● ● ●
Pheasants/quail/ grouse/ turkeys	● ●	● ● ●	● ● ●
Common peafowl, guinea fowl	● ●	● ● ●	● ●
Doves/pigeons	○	b	● ● ●
Songbirds	● ●	● ●	● ● ●
Hawks/owls	○	●	● ●
Penguins	○	○	● ●
Canaries/parrots parakeets	● ●	●	● ● ●

● ● ● Frequent
● ● Occasional
● Infrequent
○ Not reported
a Ducklings are susceptible, but the disease rarely occurs; geese are not susceptible.
b Pigeons are highly resistant to this disease.

of the small intestine, the caecum, and the colon. As the disease progresses, the intestinal lining becomes coated with a pale, tightly adhering, fibrinous material. The intestinal caecum contains thick crumbly necrotic cores in some infected birds. Enlargement and impaction of the rectum are reported as common findings in domestic ducklings. Arthritis is a common finding in the wings of pigeons. Domestic ducks with paratyphoid infections often have arthritis of the hips and knee joints.

Diagnosis

Submit whole carcasses for examination. Salmonellosis can be diagnosed only by laboratory isolation and identification of *Salmonella* spp. from infected tissues in conjunction with pathological findings because gross lesions of salmonellosis can be similar to other diseases, including avian cholera and colibacillosis. Birds with markedly abnormal behavior patterns (convulsions, tumbling) often have lesions observable by microscopic examination of the brain. Isolation of salmonellae from an intestine without significant lesions accompanied by isolation of the bacteria from other tissues generally indicates that the bird was a carrier rather than a victim of salmonellosis.

When whole carcasses cannot be submitted, remove the liver, heart, and intestine, and wrap each different tissue in a separate piece of aluminum foil. Place the foil-wrapped specimens in tightly sealed plastic bags, and ship them frozen to the diagnostic laboratory (see chapters on Specimen Collection and Preservation and on Specimen Shipment).

Fecal droppings can be checked for *Salmonella* spp., but these need special handling and should not be submitted as diagnostic specimens without prior discussions with the diagnostic laboratory. Submission of whole eggs should be considered when low hatchability is encountered. Egg shells and shell membranes can also be cultured for salmonellae. This is an effective means of detecting salmonellae in eggs that have hatched, provided the egg fragments have not been subjected to environmental conditions that would destroy the bacteria. Eggs, too, should only be submitted following consultation with disease specialists.

Control

Despite the virtually universal distribution of *Salmonella* spp., control of salmonellosis is generally not required in free-living migratory birds because the clinical forms of this disease show low prevalence in these species. However, point sources of contamination can be the cause of acute outbreaks where large numbers of birds become infected and die (Fig. 9.2). Aviaries and bird feeding stations have been the sites of major losses from paratyphoid infections.

Disease control at bird feeders should be encouraged among the public by educating people to maintain clean feeders and remove spilled and soiled feed from the area under the feeder. Occasional disinfection of the feeder with household bleach is recommended. In the event of a die-off from salmonellosis, more rigorous disinfection of feeding stations is necessary, and station use should be discontinued temporarily.

Other potential point sources of infection include refuse, sewage wastewater, and wastewater discharges from livestock and poultry operations. The potential for contaminating migratory bird habitat with *Salmonella* spp. should be considered when wastewater is intentionally used to create wetland habitat; when existing wetlands are used to receive wastewater discharges; and when development of landfill, livestock, and poultry operations are proposed in areas where an interface with migratory birds is likely.

Control of salmonellosis in captive flocks of migratory birds is necessary to prevent major losses, especially in young birds. Control of this disease should be of continual concern whenever migratory birds and other wild birds are being propagated for release programs, or are being maintained in captivity during rehabilitation programs. Strict sanitation measures need to be instituted and judiciously followed. *Salmonella* carriers should be identified by fecal culturing and should be destroyed. Multiple periodic fecal cultures are required to identify carrier birds because salmonellae are intermittently shed from the intestine. Carrier status among migratory birds is most common in waterfowl, gulls, and seabirds.

Infected adults should never be used for breeding. Antibiotic therapy may aid in overcoming an outbreak of salmonellosis, but will not eliminate car-

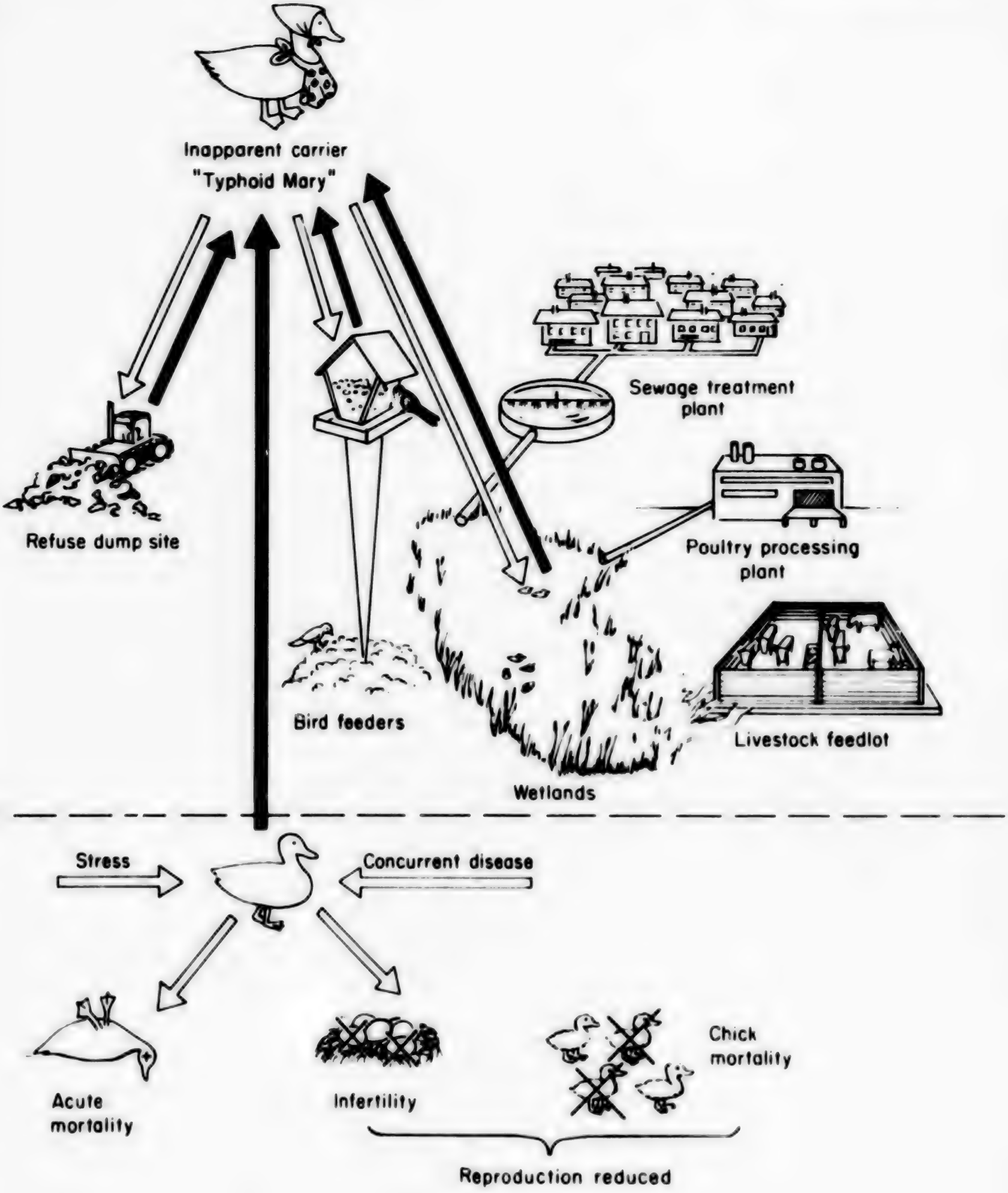

Fig. 9.2. *(a)* Sources and *(b)* consequences of salmonellosis in wild birds.

riers. Storage of food in rodent- and insect-proof containers should also be part of a prevention program. Many outbreaks in domestic poultry operations have been traced to food contaminated by rodent feces. Rats and mice are common sources of salmonellae.

Human Health Considerations

Bacteria of the genus *Salmonella* are well documented as human pathogens. "Food poisoning," characterized by acute intestinal pain and diarrhea, is the most common form of human infection. However, more serious forms of salmonellosis also occur in humans. Although the general level of *Salmonella* spp. in most species of wild birds is low, preventing infections depends on extra care with personal hygiene by personnel handling these birds or materials soiled by bird feces.

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Chapter 10.
Chlamydiosis

Louis N. Locke

Synonyms

Parrot fever, psittacosis, ornithosis, parrot disease, Louisiana pneumonitis

Cause

Chlamydiosis is caused by a unique group of infectious agents that are a link between the viruses and bacteria. These intracellular parasites grow only within the cytoplasm of living cells and for classification purposes are considered rickettsialike. *Chlamydia psittaci* is the species generally associated with this disease in birds. Direct contact with infected birds and inhalation of the causative organism in airborne particles are the usual routes of transmission for this disease. Large numbers are expelled in the feces of infected bird, and dried infected bird feces at bird roosts are especially hazardous.

Species Affected

Chlamydial infections have been found in a wide variety of mammals and birds (Fig. 10.1). This chapter focuses on the disease in birds. Although first recognized as an infectious disease affecting parrots, parakeets, and persons involved in the international parrot trade in the late 1920's to 1930's, chlamydiosis has since been recognized as a serious disease of domestic turkeys in the United States, of domestic ducks and geese in central Europe, and as a common worldwide infection of domestic and feral pigeons. The rock dove (feral pigeon) in cities is the most common and consistent source of *Chlamydia*.

Chlamydial infections have been reported from 140 species of wild birds in 15 orders. The majority of all isolations have been made from six groups of birds (Fig. 10.2). Chlamydiosis is also the occasional cause of deaths of egrets, northern fulmars, and gulls. Waterfowl, herons, and rock doves are the most commonly infected wild birds in North America (Fig. 10.3).

Distribution

Chlamydiosis has been found worldwide in feral rock doves, in gulls and northern fulmars on islands of coastal Great Britain, in waterfowl and shorebirds on the Caspian Sea, and in herons and waterfowl throughout the United States (Fig. 10.4). Infected parrots and parakeets have been found throughout the tropics and Australia.

Seasonality

Young birds are more susceptible than adults and rapid spread of the disease can occur among colonial birds during nesting. Individual cases can occur at any time.

Field Signs

Chlamydial infections in birds can be acute, subacute, or inapparent. Cases in wild birds are usually latent, the infected birds usually serving as asymptomatic carriers.

The acute form is often rapidly fatal. The bird becomes weak, stops eating, and develops pussy exudate of the eyes and nostrils. Birds tend to become

Fig. 10.1. Relative occurrence of chlamydial infections in animal groups (in descending order).[a]

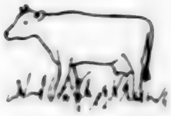
Pet birds (psittacines, finches, and pigeons)

Domestic fowl (turkeys, geese, and ducks)

Feral pigeons in cities

Wild birds (psittacines, waterfowl, and egrets)

Domestic or semi-domestic mammals (cats, goats, sheep, cattle, laboratory animals)



Wild mammals (rodents, rabbits, and opossums)

Ectoparasites (mites, chiggers, and ticks from wild or domestic animals)

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[a] Adapted from Page, 1971

Fig. 10.2. Relative occurrence of reported chlamydiosis in the most frequently infected groups of wild birds. [a]

Type of bird	Number of species infected	
	Free-living birds	Captive birds
Parrots	6	31
Songbirds	16	14
Shorebirds, gulls, alcids	23	–
Waterfowl	16	–
Herons and allies	7	–
Pigeons and doves	5	1

[a] Adapted from Page, 1971

Fig. 10.3. Relative occurrence of chlamydial infections in wild birds in North America.

Type of bird	Frequency of occurrence
Waterfowl	● ● ●
Cranes	○
Herons, egrets, ibises	● ● ●
Shorebirds	●
Gulls, terns	● ●
Raptors	○
Upland game birds	●
Feral pigeons	● ● ● ●
Songbirds	●

● ● ● ●	Common
● ● ●	Frequent
● ●	Occasional
●	Infrequent
○	Rare or not reported

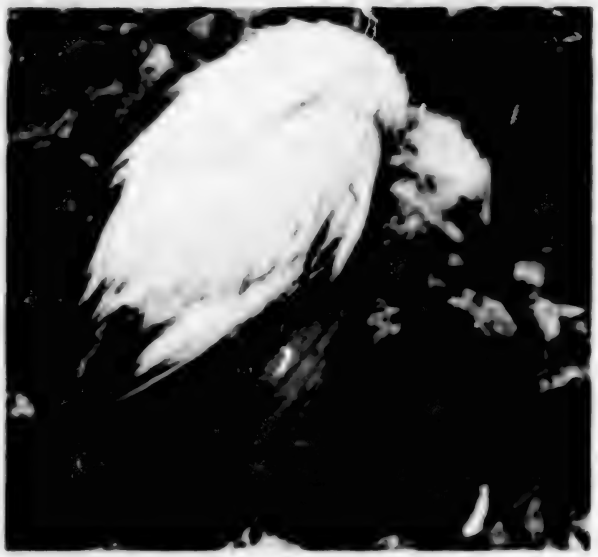


Fig. 10.4. Classical appearance of an immature little blue heron with severe chlamydial infection. *M.F.*

motionless and remain in a fixed position, huddled up with ruffled feathers. Diarrhea is often marked, and feces are frequently grayish to rust-colored, the latter the result of being tinged with blood. Respiratory distress is common.

Although no outbreaks of chlamydiosis resulting in sick or dead birds have been reported in wild waterfowl, serious outbreaks have occurred in domestic flocks of ducks and geese in central Europe. The following descriptions are based on those reports.

Sick ducks are weak, often exhibit an unbalanced gait, and become very thin. There is usually a pussy discharge from the eyes and nostrils; eyelids are *often* swollen shut ("glued together") and feathers around the eyes matted down by the discharges. There is usually a light green to grayish green to light brownish watery diarrhea. Serious losses (20% to 60–70%) of young ducklings (2 to 4 weeks old) have occurred on commercial duck farms, and the disease has been recognized in a 4-day-old duckling.

Captive snowy and great egrets having chlamydiosis exhibited weakness, abnormal gait, ruffled feathers, diarrhea, and rapid weight loss. Death often occurred 1 to 2 days after the onset of symptoms. In other egrets, the infection has been inapparent.

Feral rock doves exhibit many of the same signs, except diarrhea is apt to be more brownish and frequently tinged with blood. Losses in squabs are often very high. The occurrence of pussy discharges from the eyes of a sick pigeon should cause the observer to think *first* of chlamydiosis.

"Sudden death" (birds dropping dead without previously observed signs of illness) has been reported among captive cage birds (Java sparrows, parrots) and among wild parrots in Australia, where King parrots were reported to "fall out of trees and die within minutes."

Gross Lesions

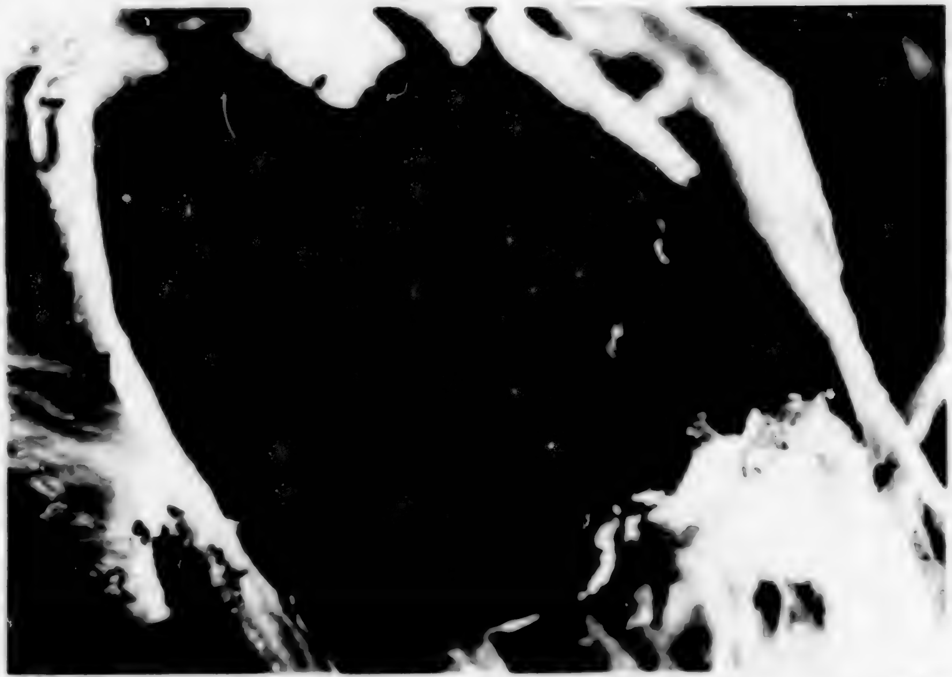
The most common anatomical change in infected birds is an enlargement of the spleen, usually to 3 or 4 times normal size. This is often seen in the inapparent carriers of the disease as well as in birds that are clinically ill.

The most striking lesions indicative of an acute or subacute infection are thick accumulations of a clear to cloudy jellylike clot of serum exudate on the heart sac, outer wall of the heart (Fig. 10.5*a*), and on the air sac membranes. Lungs are often redder than normal. The liver is often enlarged, it can show areas of necrosis, and the liver capsule is thickened (Fig. 10.5*b*).

Diagnosis

Diagnosis is based on the isolation of *Chlamydia* spp. from tissues of infected birds. Whole birds should be submitted. When this is not possible, selected tissues should be collected (see chapters on Specimen Collection and Preservation and Specimen Shipment). Lung, spleen, liver, and affected air sacs are the preferred tissues for microbial examination.

Diagnosis cannot be based on gross lesions alone since some other diseases have similar signs. Chronic avian cholera infection can produce similar gross lesions in gulls, avian malarias can cause enlarged spleens, and early stages of aspergillosis can produce somewhat similar changes in lungs and air sacs.



10.5a

10.5b

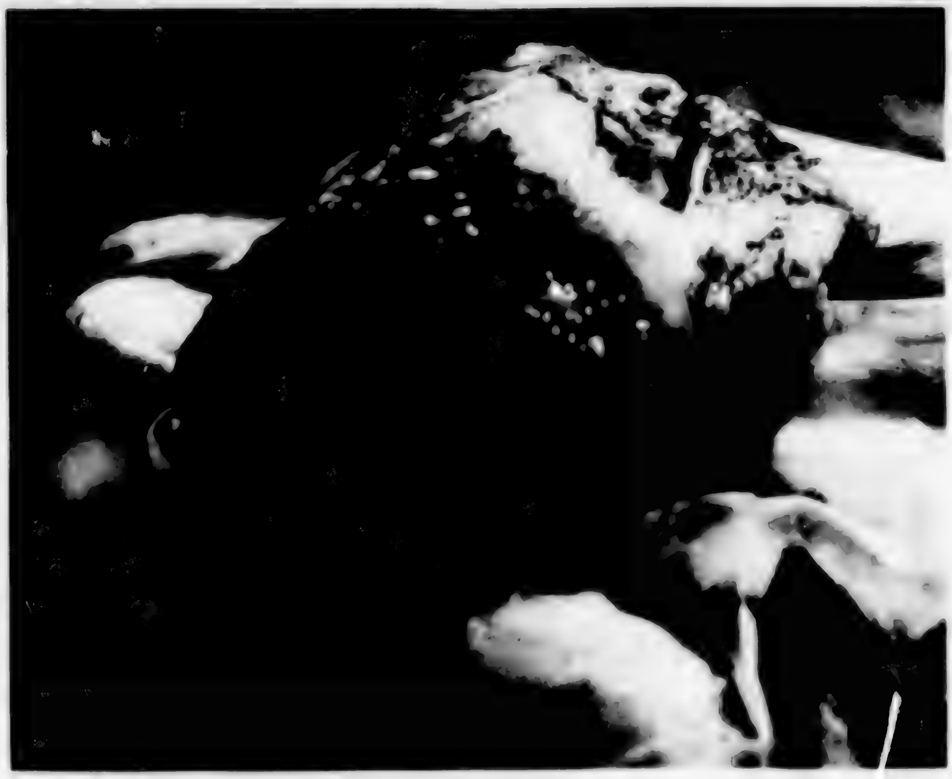


Fig. 10.5. Typical lesions of chlamydiosis in birds. (*a*) Exudate and thickening of liver capsule. (*b*) Thick exudate on pericardium. *M.F.*

Control

Chlamydia are present in the diarrhetic feces and in the lacrimal and nasal discharges of infected birds; when the excreta and discharges dry, the resulting material can become airborne. Infection usually occurs as the result of inhaling dried, fecal material that contains the infectious *Chlamydia* organisms.

Individuals working in areas where there is a strong possibility of inhaling airborne avian fecal material should consider wearing a mask or respirator. Dry, dusty areas having bird droppings should be wetted down with disinfectants such as commercial bleaches or Environ. Insofar as possible, avoid working with large numbers of birds in dusty, closely confined areas.

Human Health Considerations

Chlamydiosis can be a serious human health problem, particularly to those working with birds. The correlation between parrots and this disease in man prompted the United States and most nations of western Europe to outlaw the importation of parrots and parakeets from 1930 to 1960.

Serious outbreaks have occurred among poultry slaughterhouse workers, and several severe cases also have occurred among wildlife biologists. These biologists were thought to have become infected from handling snow geese, great egrets, snowy egrets, white-winged doves, and ducks.

Before antibiotics were available, chlamydiosis had a case fatality rate of about 20%. Today, such fatalities are rare. Nevertheless, biologists working with birds should acquaint their physicians with their duties so that early signs of chlamydiosis will not be dismissed as "a case of the flu" or some other general malady.

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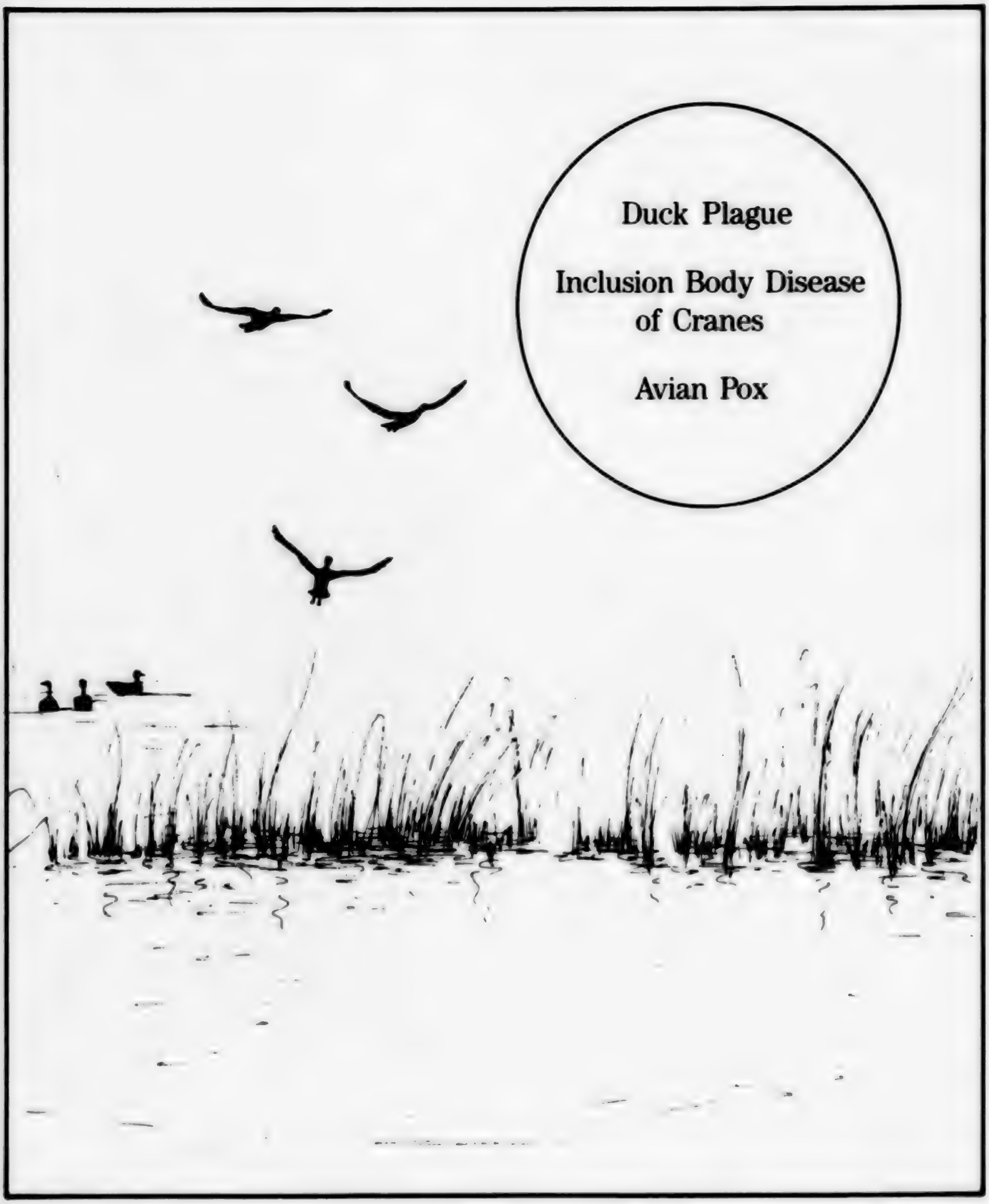
Viral Diseases



Duck Plague

Inclusion Body Disease of Cranes

Avian Pox

Chapter 11.
Duck Plague

Christopher J. Brand

Synonyms

Duck virus enteritis, DVE

Cause

Duck plague is an acute, contagious, and often fatal disease caused by a herpesvirus. As with many other herpesviruses, duck plague virus can establish inapparent infections in birds surviving exposure, a state in which the virus cannot be detected by standard methods for virus isolation. Outbreaks are thought to be primarily a result of virus shedding by carrier birds, which causes infection and disease among susceptible waterfowl. Bird-to-bird contact and contact with virus that has contaminated the environment perpetuate an outbreak.

Species Affected

Only ducks, geese, and swans are susceptible to duck plague. American coots, grebes, shorebirds, cranes, and other aquatic birds do not become infected. Susceptibility varies a great deal among waterfowl species (Fig. 11.1). In one study it took 300,000 times more virus material to infect northern pintails than blue-winged teal.

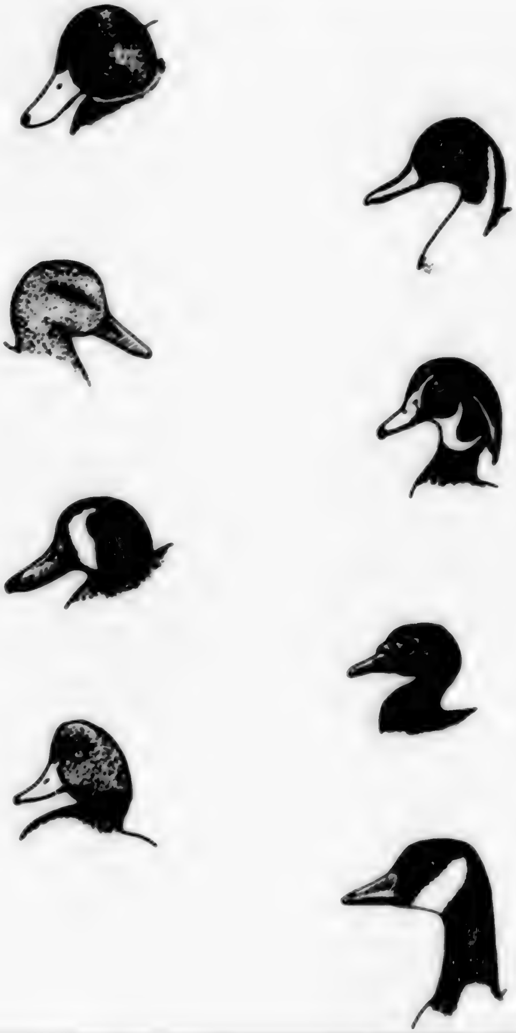
Distribution

Duck plague was not known to occur in North America until a 1967 outbreak in the white Pekin duck industry on Long Island, New York. Since then, outbreaks of duck plague have occurred from coast to coast and from Canada to Texas (Fig. 11.2). The disease has also been reported in several European countries since it was first recognized in the Netherlands in 1923 and in Asia in 1958 (Fig. 11.3).

Despite the widespread geographic distribution and frequent occurrence of duck plague in captive waterfowl in North America, wild waterfowl have been involved only infrequently. The only major outbreak in migratory waterfowl occurred in January 1973 at Lake Andes National Wildlife Refuge, South Dakota, when more than 40,000 of 100,000 mallards and a smaller number of Canada geese and other species died while wintering there (Fig. 11.4). During the initial 1967 outbreak in white Pekin ducks on Long Island, several hundred wild waterfowl (primarily mallards and American black ducks) also died on adjacent Flanders Bay, apparently as a result of spillover of the disease in white Pekin ducks. With the exception of the Lake Andes and Flanders Bay outbreaks, involvement of migratory waterfowl has been limited to a small number of birds. All confirmed outbreaks have also involved commercial, avicultural, captive-raised, or feral (city park-type) waterfowl.

The distribution of duck plague among free-living North American waterfowl populations is unknown. Some believe that the large numbers of surviving wild waterfowl exposed to this disease at Flanders Bay and Lake Andes became potential disease carriers, and now duck plague is widespread among migratory waterfowl. However, surveys of wild waterfowl conducted by the U.S. Department of Agriculture in 1967 and the National Wildlife Health Center (NWHC) in 1982 and 1983 did not detect any evidence of duck plague carriers. In the latter survey, more than 4,500 waterfowl across the United States were sampled (Fig. 11.5). Sampling sites included major waterfowl concentration areas and areas where duck plague has been a recurrent disease problem in captive and feral waterfowl. Although none of the birds sampled was shedding detectable duck plague virus, the problem of inapparent carriers previously described complicates interpretation of these results.

Fig. 11.1. Comparative susceptibility of eight waterfowl species to duck plague.



Type of bird	Relative susceptibility
Mallard	● ●
Pintail	●
Gadwall	● ●
Wood duck	● ● ●
Blue-winged teal	● ● ●
Muscovy	● ●
Redhead	● ● ●
Canada goose	● ●

● ● ● Highly susceptible
● ● Moderately susceptible
● Slightly susceptible

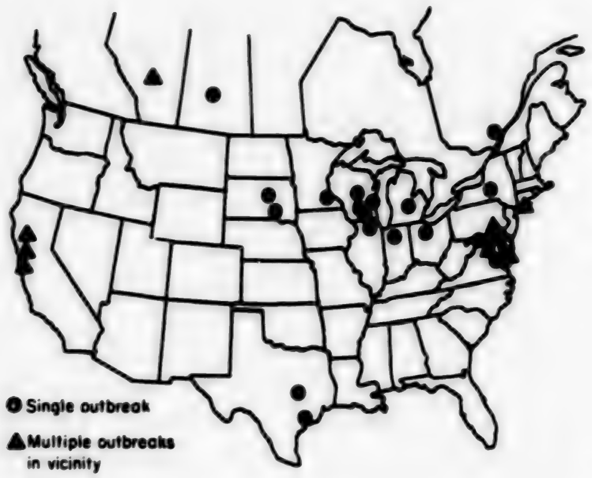


Fig. 11.2. Confirmed duck plague outbreaks in North America, 1967–85.

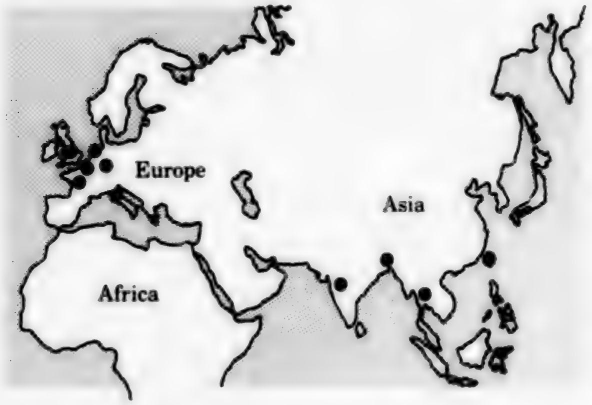


Fig. 11.3. Geographic distribution of duck plague (exclusive of North America).

The absence of this disease as a cause of mortality in the thousands of wild waterfowl necropsied by the NWHC provides additional evidence that duck plague is not an established disease in wild North American waterfowl. These examinations, performed since 1975, involve waterfowl found dead on National Wildlife Refuges and other major waterfowl concentration areas.

Seasonality

Duck plague has occurred throughout the year, although most outbreaks happen during April, May, and June (Fig. 11.6).

Field Signs

There is no prolonged illness associated with duck plague. Therefore, sick birds are seldom seen in the field and birds that are healthy 1 day may be found dead the next. The incubation period between virus exposure and death is generally 3 to 7 days in domestic ducks, and as long as 14 days in wild waterfowl (experimental studies). Wing-clipped mallards released to monitor the Lake Andes duck plague outbreak died 4 to 11 days after their release.

Sick birds may exhibit hypersensitivity to light, causing them to seek dense cover or other darkened areas. Extreme thirst, droopiness, and bloody discharge from the vent (Fig. 11.7*a*) or bill (Fig. 11.7*b*) may also occur. The ground may appear blood-stained where sick birds have rested. In males, the penis may be prolapsed (Fig. 11.7*c*). Some infected birds may have an ulcerative "cold sore" lesion under the tongue from which virus can be shed (Fig. 11.7*d*). Routine examination of apparently healthy waterfowl for this lesion during banding operations may be helpful in identifying inapparent carriers. Birds found with these lesions should be euthanized and submitted to a qualified disease diagnostic laboratory.

Death may be preceded by loss of wariness, inability to fly, and finally by a series of convulsions that could be misinterpreted as pesticide poisoning or other diseases such as avian cholera (Fig. 11.8).

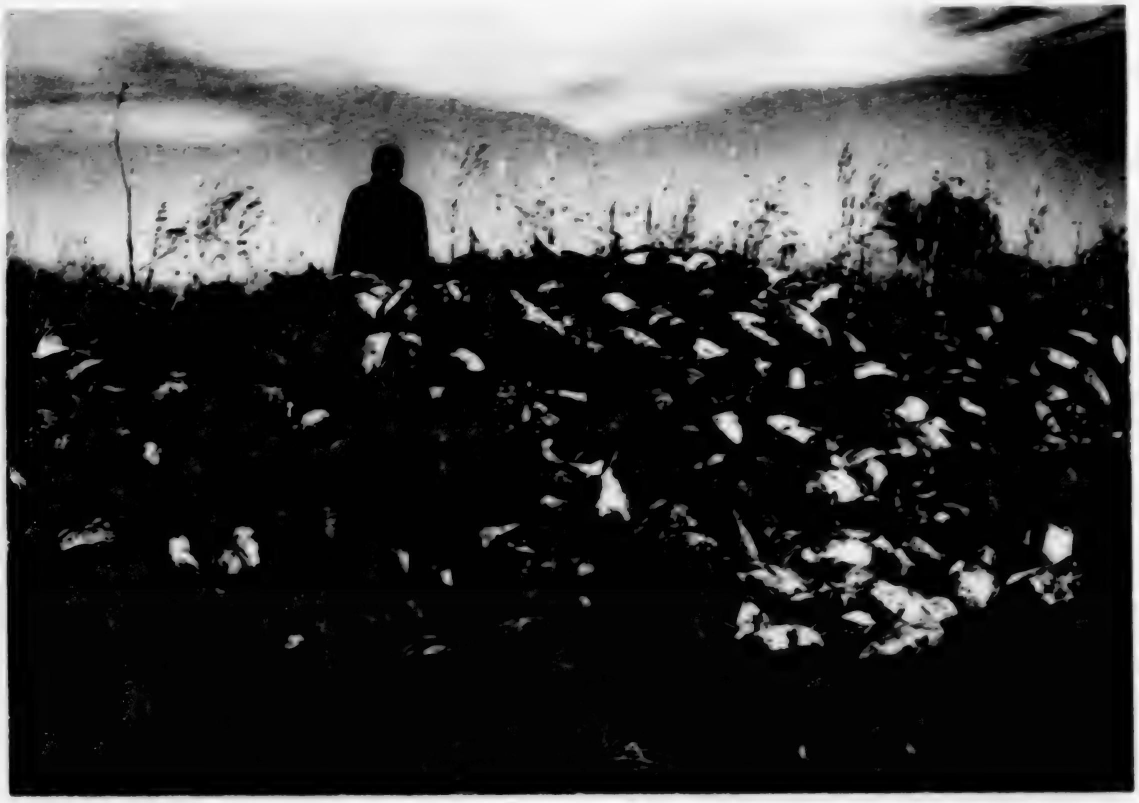


Fig. 11.4. During the 1973 outbreak of duck plague at Lake Andes National Wildlife Refuge in South Dakota, more than 40,000 mallards died. *M.F.*

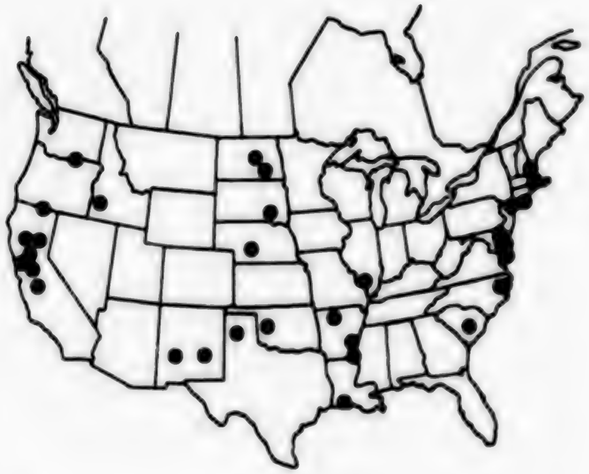


Fig. 11.5. Sampling locations for 1982–83 duck plague survey.

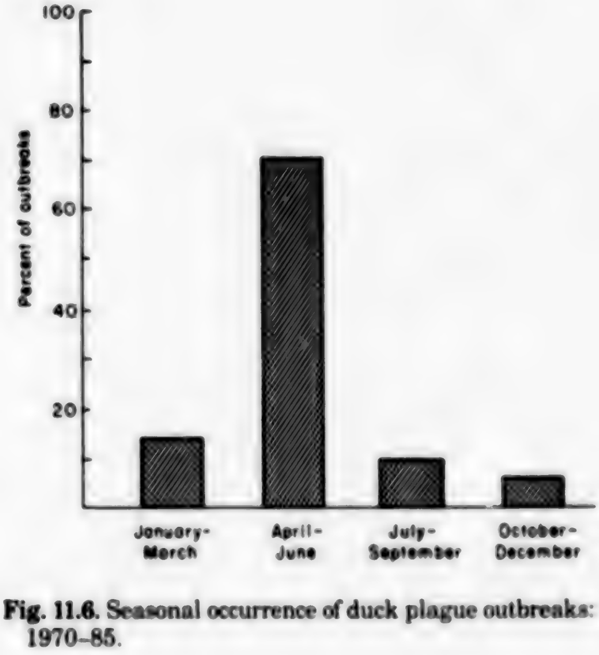
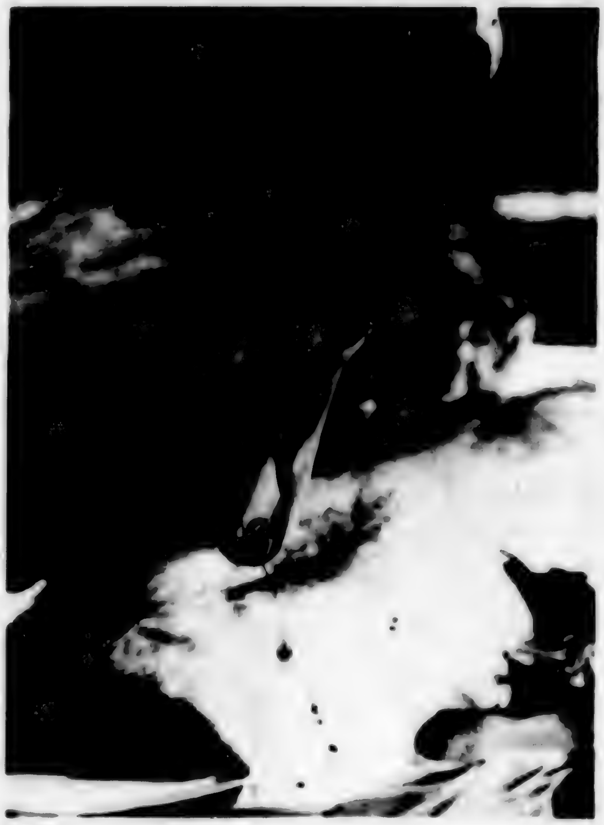
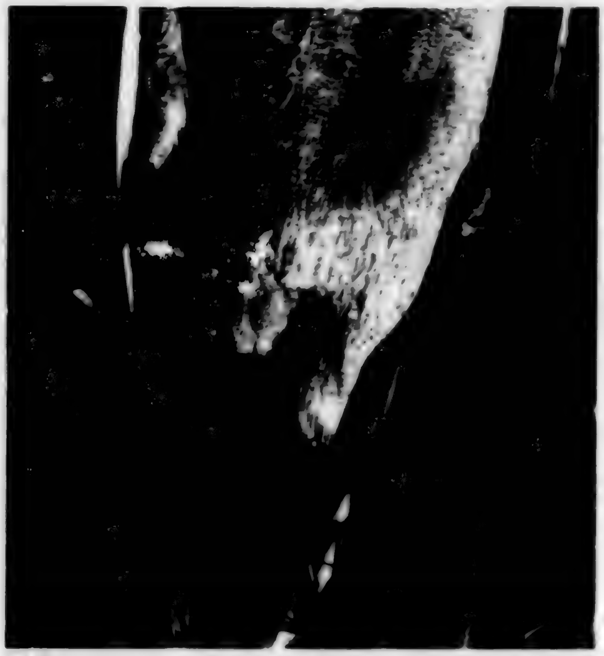


Fig. 11.6. Seasonal occurrence of duck plague outbreaks: 1970–85.

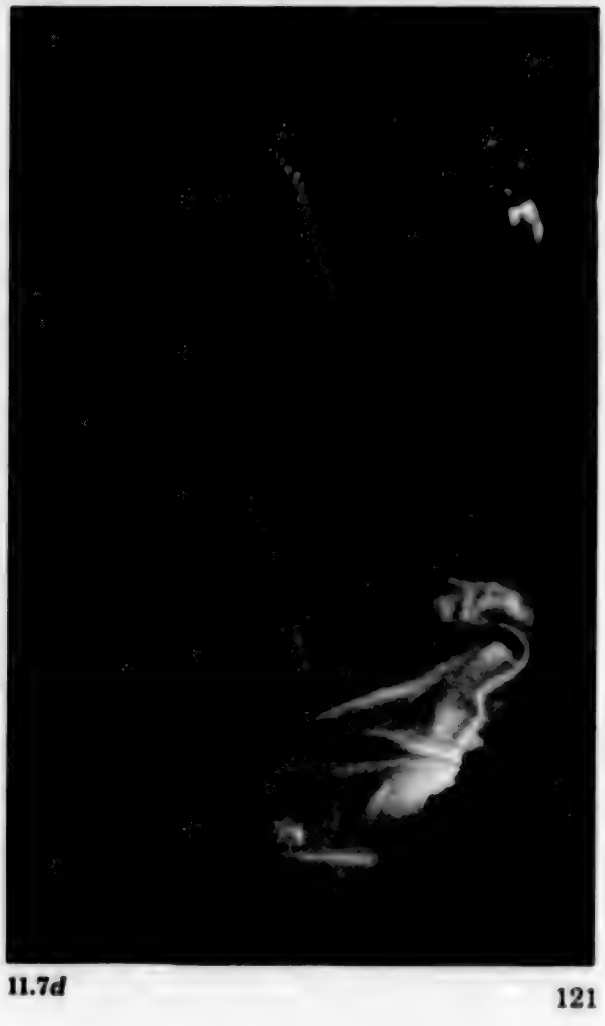
11.7*a*



11.7*b*

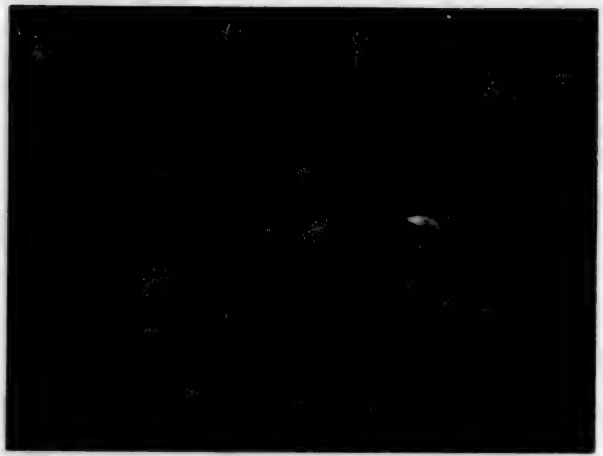


11.7*c*



11.7*d*

Fig. 11.7. Field signs associated with duck plague include: (*a*) blood staining of the vent area, (*b*) blood dripping from bill or blood-stained bill, (*c*) prolapse of the penis, and (*d*) a "cold sore" under the tongue. (*a*, *b*, and *c*) *M.F.*; (*d*) *J.R.*



11.8a

11.8b

11.8c

11.8d

Fig. 11.8. Death sequences observed during terminal stages of duck plague infection at Lake Andes National Wildlife Refuge began with (*a*) the head of the bird dropping forward, wings becoming partially extended from the sides, and the tail becoming fanned and rigid. This was followed by (*b*) the bird swimming in a tight circle while rapidly beating the water with its wings and with the head pulled back and twisted to the side. (*c*) At times birds would fall over on their side, be unable to regain a normal body position, and drown. (*d*) Other birds would simply stop swimming, relax, and quietly die. This entire sequence generally lasted only a few minutes. *M.F.*

Gross Lesions

Duck plague virus attacks the vascular system, resulting in hemorrhaging and free blood throughout the gastrointestinal tract (Fig. 11.9*a*). At the Lake Andes outbreak, the most prominent lesions were hemorrhagic or necrotic bands circumscribing the intestine in mallards (Figs. 11.9*b*, *c*, and *d*) and disk-shaped ulcers in Canada geese (Figs. 11.9*e* and *f*). Sometimes there were "cheesy," raised plaques along the longitudinal folds of the esophagus and proventriculus (Fig. 11.10*a*) and on the mucosal surface of the lower intestine (Fig. 11.10*b*). Areas of tissue death (spots) were also evident in the liver (Fig. 11.10*c*), as was hemorrhaging on the heart surface of some birds (Fig. 11.10*d*).

It is important to recognize that not all lesions occur in all birds at all times. Outbreaks of duck plague in captive and semi-captive waterfowl have often resulted in infected birds with less distinct lesions. Of all the lesions illustrated, those of greatest value in diagnosing duck plague are hemorrhagic or necrotic bands or disks within the intestine, large amounts of free blood in the digestive tract, and cheesy plaques in the esophagus and cloaca. Liver and heart lesions of duck plague are grossly similar to those of avian cholera and cannot be used to distinguish between these two diseases.

Diagnosis

Although a presumptive diagnosis of duck plague may be made on basis of characteristic internal lesions, final diagnosis can only be made by virus isolation and identification. Because of the threat that duck plague poses, ducks, geese, and swans exhibiting characteristic signs or lesions should be euthanized and shipped to a qualified diagnostic laboratory as quickly as possible. Whole birds rather than tissues should be submitted. When this is not possible, the liver should be removed, wrapped in clean aluminum foil, and then placed in a plastic bag and frozen for shipment. Take particular care in preserving and packaging specimens to avoid their decomposition during transit and contamination of the shipping containers (see chapters on Specimen Collection and Preservation and Specimen Shipment).

Control

Birds with inapparent duck plague infections are probably the major reservoir of this disease and pose the greatest problem for disease prevention and control. Clinically ill birds actively shed the virus and are recognized as sick birds. However, asymptomatic healthy duck plague carriers can shed the virus periodically for life but are not overtly identifiable. As with other herpesvirus infections, stress caused by factors such as reproduction and egg laying and inclement weather appears to play an important role in inducing virus shedding.

Virus particles are shed by infected birds through feces and oral discharges, resulting in contamination of food and water. Scavenging and decomposition of carcasses also contaminate the environment by releasing viruses from tissues and body fluids. Virus transmission through the egg has been reported, but the role of the egg in the disease cycle remains to be resolved.

Control of duck plague outbreaks requires rapid response and agressive actions to prevent the spread and establishment of this disease. The close association between duck plague outbreaks and captive waterfowl, especially mallards, needs to be considered. Waterfowl release programs should not use birds or eggs from flocks with a history of this disease. Birds scheduled for release should be maintained in confinement for at least 2 weeks before release. Birds that die during this period should be submitted to a qualified disease diagnostic laboratory.

Destruction of infected flocks, including eggs, is recommended whenever possible because infected birds that survive are likely to become carriers and can initiate subsequent outbreaks. Presently, there are no effective means of determining whether there are carriers in a flock because the virus cannot be detected in inapparent carriers by standard laboratory methods. Also, blood testing is ineffective for detecting birds that survived previous exposure to this disease because the presence of detectable antibody is short-lived and unpredictable. New techniques now being developed provide promise for overcoming this difficulty.

Duck plague virus is hardy, and can remain viable for weeks under certain environmental conditions. For example, the virus could be recovered from Lake Andes water held at 4°C for 60 days under laboratory conditions. Duck plague virus is

11.9a

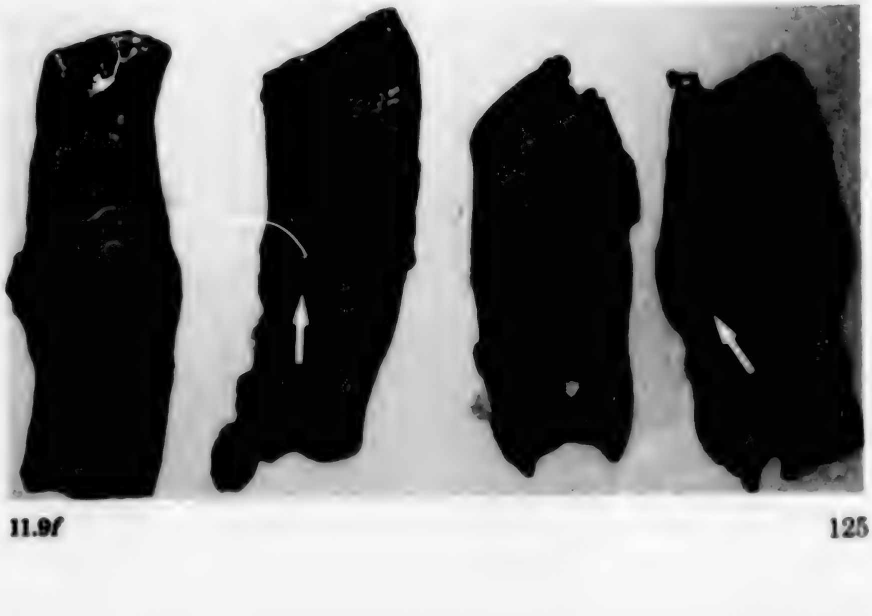
11.9b

11.9c

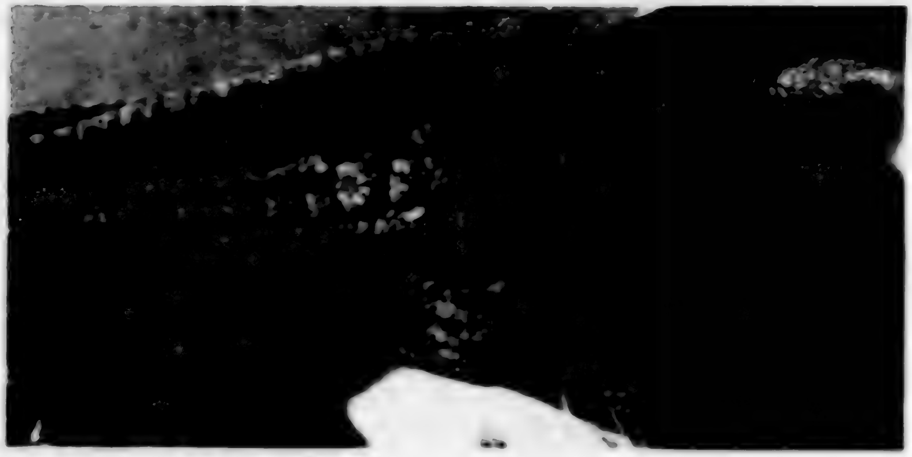
11.9d

11.9e

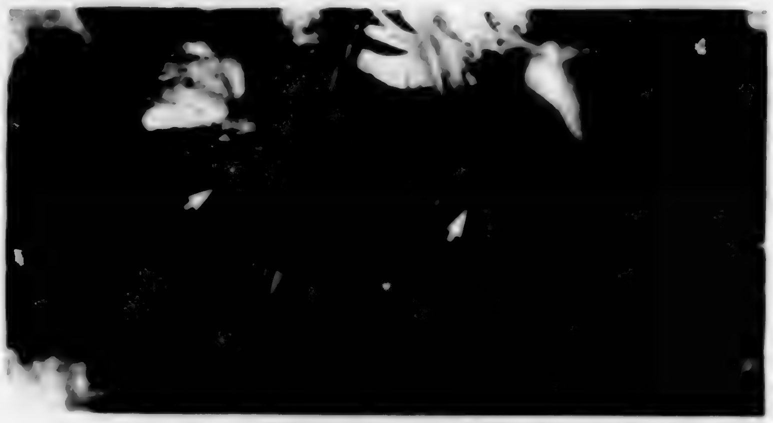
Fig. 11.9. Appearance of major lesions of duck plague: (*a*) hemorrhage and free blood in lumen of intestine; (*b* and *c*) external appearance of bands of necrotic tissue in mallard intestine; and (*d*) appearance of bands when intestine is opened; (*e*) external appearance of similar lesions in intestine of a Canada goose; and (*f*) buttonlike rather than bandlike appearance of lesions when intestine is opened. (*a,b,c,e,* and *f*) *M.F.*; (*d*) *J.R.*



11.9f



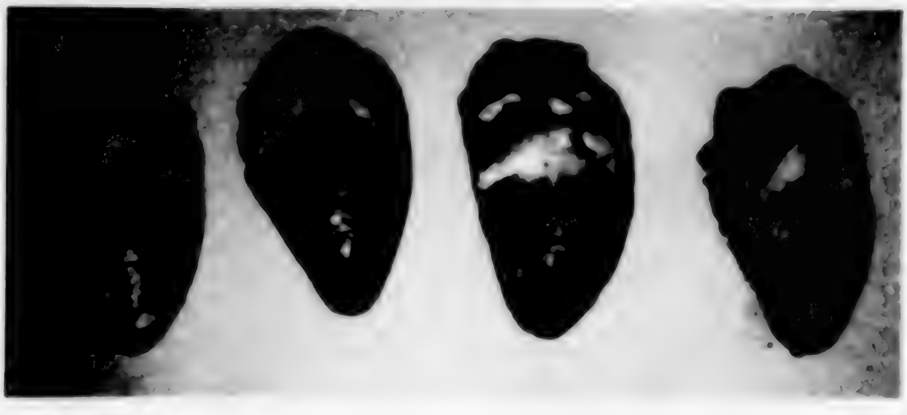
11.10a



11.10b

11.10c

Fig. 11.10. Other internal lesions of duck plague include: (*a*) cheesy, raised plaques along the longitudinal folds of the esophagus, proventriculus, and (*b*) inside (mucosal) surface of the lower intestine. (*c*) Necrotic spots may occur in the liver and (*d*) hemorrhages on the heart surface. (*a*) *S.S.*; (*b,c* and *d*) *M.F.*



11.10d

instantly inactivated at pH 3 and below and pH 11 and above. Therefore, rigorous decontamination of infected waters (e.g., by chlorination) and grounds (i.e., by raising pH); and burning or decontamination of physical structures, litter, and other materials at outbreak sites should be carried out to the extent possible. Carcass collection should be thorough and incineration used for disposal. Personnel and equipment used at outbreak sites should be decontaminated before leaving the site to prevent mechanical spread of the virus to other waterfowl areas. Environ and chlorine bleach are basic disinfectants suitable for this purpose (see chapter on Disease Control Operations).

An attenuated live-virus vaccine has been developed for combating duck plague in domestic white Pekins, but this vaccine has not been proven entirely reliable in protecting other species of ducks and geese. It should not be considered as a means of controlling or preventing outbreaks in migratory birds.

Human Health Consideration

None.

Supplementary Reading

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Chapter 12.
Inclusion Body Disease of Cranes

Douglas E. Docherty

Synonym

Crane herpes

Cause

In 1978 a previously unidentified herpesvirus was isolated at the National Wildlife Health Center (NWHC) from a die-off of cranes in a captive population. Serological testing of this virus against other previously isolated avian herpesviruses did not result in cross-reactions, thereby supporting this agent's status as a distinctly new virus. The NWHC assigned the descriptive name of inclusion body disease of cranes (IBDC) to this disease when reporting the outbreak in the scientific literature.

Very little is known about how this disease is transmitted. As with duck plague and avian cholera, outbreaks are thought to be initiated by disease carriers within a population of birds. Spread of the disease probably occurs as a result of direct contact between infected birds and other susceptible birds and by contact with a virus-contaminated environment.

Species Affected

Spontaneous infections have occurred in several species of captive cranes whose ages ranged from immature to adult (Fig. 12.1). Laboratory-induced infections and death occurred in adult cranes and in white Pekin ducklings between 3 and 17 days old, but not in 64-day-old Muscovy ducks. Adult American coots were also susceptible, but white leghorn chicks were not (Fig. 12.2). These findings suggest that at least several species of cranes can become infected by this virus (virus replication occurs in the bird following exposure), but the occurrence of illness and death is highly variable among different crane species. Too little is known about this disease to assess susceptibility in other species.

Distribution

The first outbreak of IBDC in the United States occurred among captive cranes at the International Crane Foundation near Baraboo, Wisconsin, in March 1978. A crane die-off also associated with a herpesvirus occurred during the winter of 1973–74 in Vienna, Austria. The relationship between these two herpesviruses has not been determined; however, the lesions and general pathological findings were similar.

Since 1978, many zoological and scientific collections have submitted crane sera for testing. Nine of these collections contained exposed (antibody-positive) cranes (Fig. 12.3). Testing of endangered species of cranes being imported into the United States has detected four additional exposed cranes. All came from Asia. Serological testing by the NWHC revealed antibody to IBDC in 11.3% of 452 samples from 14 species of captive cranes. Results from other laboratories are not available because of confidentiality considerations. However, it is known that some antibody-positive cranes have been detected in these collections in addition to those detected by the NWHC.

There is no evidence that wild North American crane populations have been exposed to IBDC. None of 95 sandhill crane sera collected in Wisconsin and Indiana during 1976 and 1977 had antibody to this virus. Additional testing is needed to further evaluate the status of IBDC in wild cranes.

Seasonality

There have not been enough known outbreaks of IBDC to indicate seasonal trends. The outbreak in Wisconsin occurred in March, and the die-off from suspected IBDC in Vienna, Austria, began in December.

Fig. 12.1. Results of natural exposure to IBDC at the International Crane Foundation, Baraboo, Wisconsin.

Cranes	Response	
	Mortality	Antibody
Stanley	●	△
Sandhill	●	▲
Manchurian	●	▲
Hooded	●	▲
Sarus	○	▲
Common	○	▲
White-Naped	○	△
Demoiselle	○	▲
Brolga	○	△
East African Crowned	○	▲

● death occurred
○ no death
▲ antibody positive
△ no antibody

Field Signs

During the 1978 die-off, signs such as lethargy and loss of appetite persisted for 48 hours, with diarrhea that was occasionally bloody just before death. Critically ill cranes died when handled.

Gross Lesions

Cranes that died from IBDC at the International Crane Foundation had swollen livers and spleens. These organs contained numerous pinpoint- to pinhead-sized lesions that appeared as yellow-white spots throughout the tissue (Fig. 12.4). Other noticeable gross lesions included hemorrhages in the thymus gland and intestines. The acute nature of the disease was evident by an abundance of subcutaneous fat in carcasses examined.

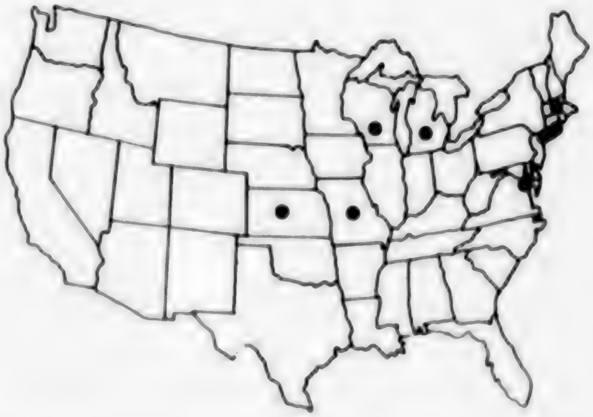


Fig. 12.3. Reported North American geographic distribution in cranes known to be exposed to the virus of IBDC.

Fig. 12.2. Known susceptibility of avian species to experimental infection of IBDC.

Type of bird		Mortality
Ducks		
White Pekin		
3 days old		●
17 days old		●
Muscovy		
64 days old		○
Coots		
Adults		●
Cranes		
Sandhill crane		●
Common crane		●
Red-crowned crane		●
Chickens		
White Leghorn		
17 days old		○

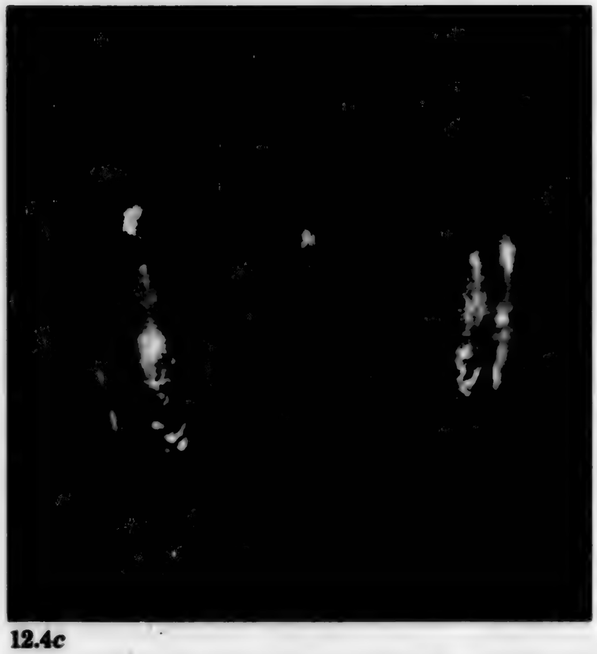
● Death
○ No death

Diagnosis

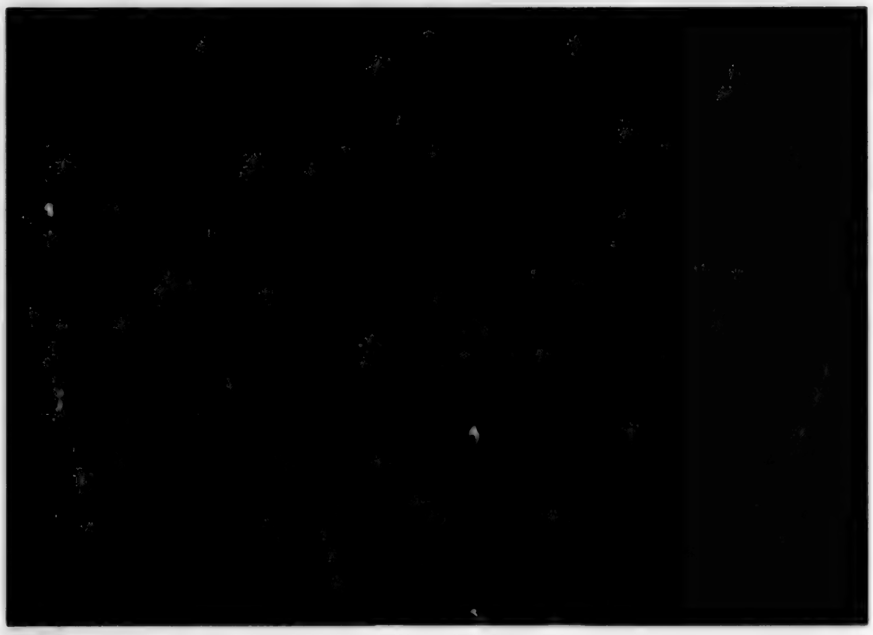
A presumptive diagnosis can be made on the basis of gross lesions in the spleen and liver (Fig. 12.4). However, laboratory confirmation of this diagnosis is essential and requires virus isolation from affected tissues. Whole carcasses should be submitted to a disease diagnostic laboratory (see chapter on Specimen Shipment). When this is not possible, the liver and spleen should be removed (see chapter on Specimen Collection and Preservation) and placed in separate plastic bags and shipped frozen. Because this disease causes characteristic microscopic lesions (intranuclear inclusion bodies in spleen and liver), it is also useful to place a piece of the liver and spleen in 10% buffered formalin when whole carcasses cannot be submitted. Care must be taken not to contaminate tissue samples being taken for virus isolation when removing a portion of these tissues for formalin fixation.

12.4*a*

12.4*b*



12.4c



12.4d

Fig. 12.4. Gross lesions of IBDC: (*a*) small, yellow-white spots throughout the cut surface of the liver; (*b*) abundance of spots create mottled appearance of liver surface; (*c*) external surface of spleen; and (*d*) cut surface of spleen. *J.R.*

Control

Any outbreak of IBDC in North America should be considered a serious event requiring immediate involvement of disease control specialists; the infected flock should be killed and the site of the outbreak decontaminated. This type of response is complicated because endangered species of cranes may be involved and such action may not be feasible. Nevertheless, failure to take aggressive action could enable IBDC to be established as a significant cause of mortality in free-living North American cranes, could jeopardize captive breeding programs for endangered species of cranes, and could result in this disease becoming a serious mortality factor in crane collections.

When captive infected flocks cannot be killed, every effort must be made to permanently isolate the survivors from other birds. Birds that survive infection can become carriers of the virus and infect other birds by intermittently discharging virus into the environment. Care must also be taken to prevent spread of the virus to susceptible birds by contact with potentially contaminated materials such as litter, water, feed, and feces from the confinement area. Clothes and body surfaces of personnel in contact with these birds are other sources of contamination.

There is no evidence for transmission of IBDC virus through the egg. However, until more is known about this disease, eggs from birds surviving infection should be disinfected and hatched elsewhere. Young from these eggs should be reared at a facility free of IBDC, tested, and found free of exposure to IBDC before the birds are allowed to have contact with other birds.

IBDC virus elicits an antibody response that persists for several years. This is a useful indicator for evidence of exposure to this virus. Birds found to have antibody to IBDC should be considered potential carriers of this virus and either be destroyed or placed in confinement under the conditions specified above.

Good husbandry practices are important in reducing the potential for transmitting IBDC and in minimizing conditions favorable to virus shedding. Crowding, inclement weather, interspecies interactions, and poor sanitation were all possible contributing factors to the die-off at the International Crane Foundation. IBDC has not reappeared at that location following initiation of corrective management actions, including isolation of survivors directly involved in the die-off, and initiation and maintenance of an aggressive flock health surveillance program.

Human Health Considerations

None.

Supplementary Reading

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Schuh, J. C. L., and T. M. Yuill. 1985. Persistence of inclusion body disease of cranes virus. J. Wildl. Dis. 21:111–119.

Chapter 13.
Avian Pox

Wallace R. Hansen

Synonyms

Fowl pox, avian diphtheria, contagious epithelioma, poxvirus infection

Cause

Avian pox is the common name for a mild to severe, slow-developing disease of birds caused by a large virus belonging to the avipoxviruses, a subgroup of poxviruses. This group contains several similar strains of virus that vary in their host specificity. Some have the ability to infect several groups or species of birds whereas others are species-specific. Mosquitos are a common means of transmitting this disease. Contact with virus-contaminated surfaces can also result in pox infections when virus enters the body through abraded skin.

Species Affected

Avian pox affects a great variety of bird species (Fig 13.1). However, reports of this disease in free-living migratory birds are limited. Only a few cases of this disease have been reported in wild waterfowl. Single occurrences have been documented in a free-flying green-winged teal in Alaska, a Canada goose in Ontario, Canada, a mallard duck in Wisconsin, a mute swan cygnet in New York, and a tundra swan in Maryland, and three cases have been reported in common goldeneye in Saskatchewan, Canada. Avian pox has also occurred in captive common scoters in the Philadelphia Zoo and in a Hawaiian goose in the Honolulu Zoo.

In 1979 a bald eagle from Alaska was diagnosed as having died from poxvirus infection. Since then, several additional cases of this disease have been diagnosed in bald eagles. Isolated cases of poxvirus infection have been reported in other wild raptors, including a golden eagle, European sparrowhawks, a red-shouldered hawk, a rough-legged hawk, and red-tailed hawks.

Large numbers of colonial nesting birds, such as the Laysan albatross, have become infected with avian pox on Midway Atoll; red-tailed tropicbirds have also been affected. Common murres rescued from an oil spill in California developed poxvirus lesions while in a rehabilitation center. Mourning doves, finches, and other perching birds using backyard feeders have been involved in a large number of epizootics. Avian pox is also suspected as a factor in the decline of forest bird populations in Hawaii and northern bobwhite in the southeastern United States.

Distribution

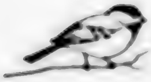
This disease occurs worldwide, but little is known about the prevalence of avian pox in wild bird populations. A few studies have indicated that poxvirus can persist at low infection rates in a population for several years. This probably accounts for the multiple reports of poxvirus infection in some species. Environmental factors such as temperature, humidity, moisture, and protective cover all play a role in the distribution of this disease. These factors affect virus survival outside of the bird host in addition to the availability of such vectors as flies and mosquitoes to feed on bird hosts.

Seasonality

Although poxvirus infections in wild birds occur during all seasons of the year (Fig. 13.2), disease outbreaks have been associated with the emergence of vector populations, environmental conditions, and the habits of the species affected. Supporting evidence from limited studies showed that the lowest disease prevalence rates for California quail in Oregon were in the dry summer months compared with the highest during fall and winter months. In Florida, reports of avian pox in wild turkeys occurred in late summer and early fall, corresponding to the mosquito season. On Sand Island

Fig. 13.1. Reported occurrence of avian pox in wild birds in North America.

Type of bird	Frequency of reports
Waterfowl	●
Wading birds	○
Shorebirds	○
Marine birds	● ● ●
Raptors	● ●
Upland game birds	● ● ●
Songbirds	● ● ●



● ● ● Multiple; reports often involve a number of birds in a single event
● ● Occasional; reports tend to involve individual birds rather than groups of birds
● Small number of reports, generally involving individual birds
○ Rare or not reported

Fig. 13.2. Seasonal outbreaks of avian pox in wild birds.

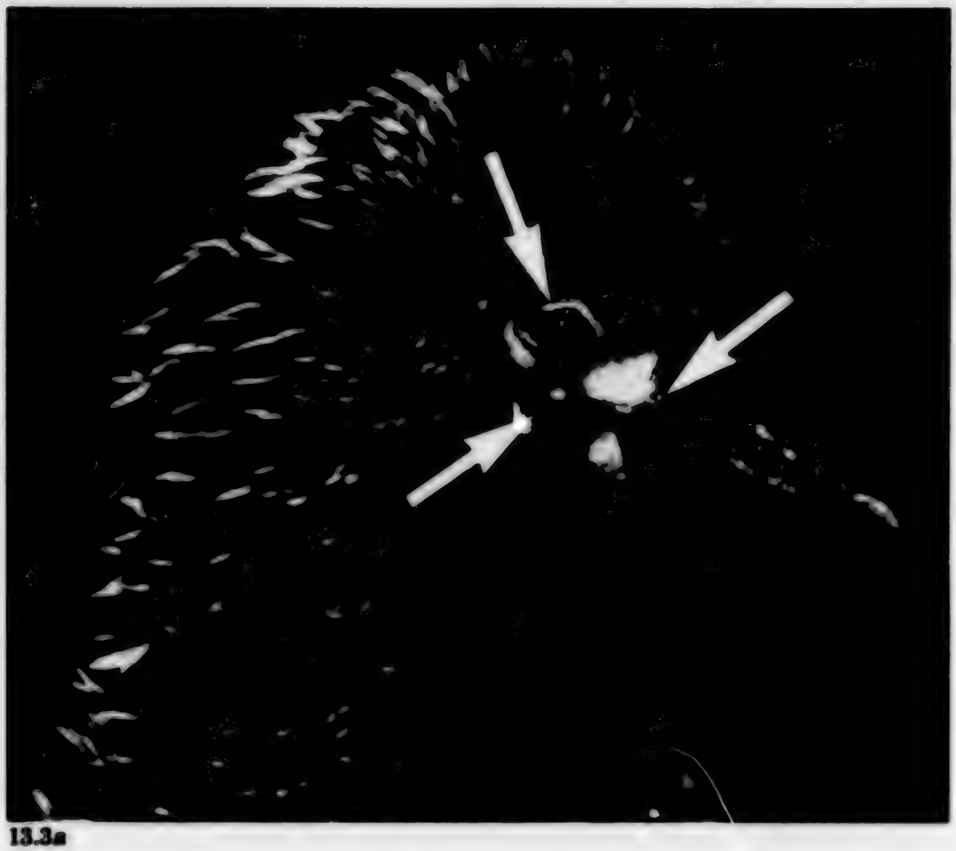
Type of bird	Season Spring	 Summer	 Fall	 Winter
Marine birds	●	○	●	
Raptors	○	○	○	○
Upland game birds		○	●	●
Songbirds	○	○		●
Hawaiian forest birds		○	●	●

● Heaviest prevalence of infection
○ Low prevalence of infection

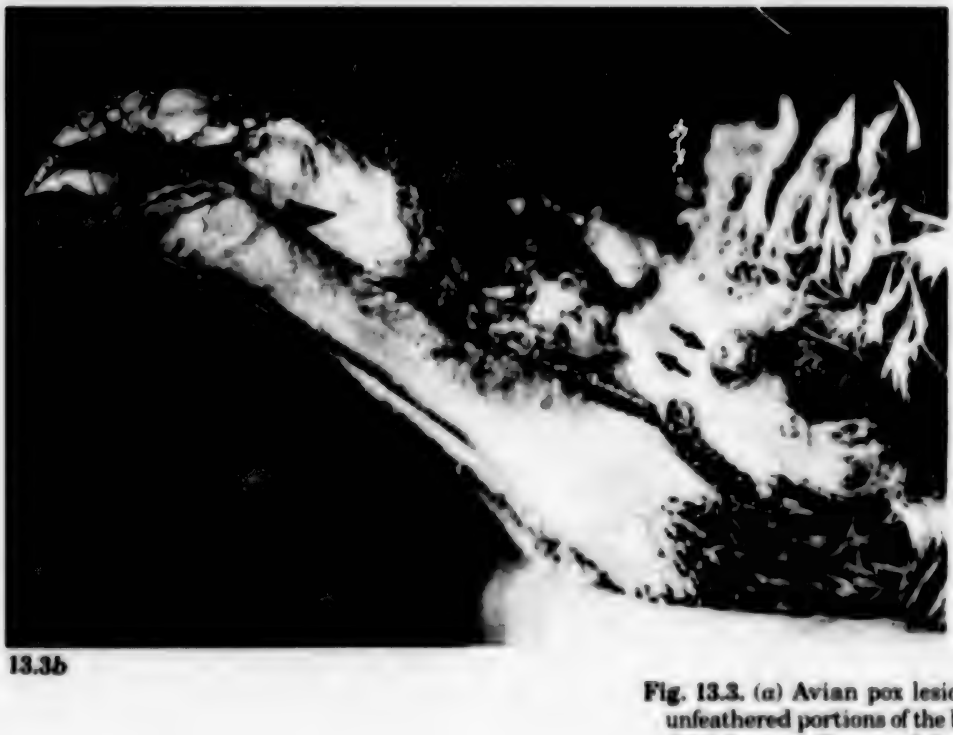
of the Midway Atoll, avian pox was first reported in September 1963 in the nestlings of the red-tailed tropicbird. In the late 1970's, the occurrence of poxvirus on the island shifted to March and April and was found in the nestlings of Laysan albatross, a species that had increased in number dramatically in recent years. It has also been noted that immature birds are usually the most frequently and severely infected with poxvirus. Bird feeding stations maintained in winter have been the prime source of several poxvirus outbreaks in the continental United States from contact transmission of the virus through close association of many bird species.

Field Signs

Birds with wartlike nodules on one or more of the featherless areas of the body, including feet, legs, base of the beak, and eye margin, should be considered suspect cases of avian pox (Fig. 13.3). The bird may appear weak and emaciated if the lesions are extensive enough to interfere with feeding. Some birds may show signs of labored breathing if air passages are partially blocked. Even though the course of this disease is long, birds with extensive lesions are known to completely recover if nutritional intake is maintained and secondary infections are avoided.



13.3a



13.3b

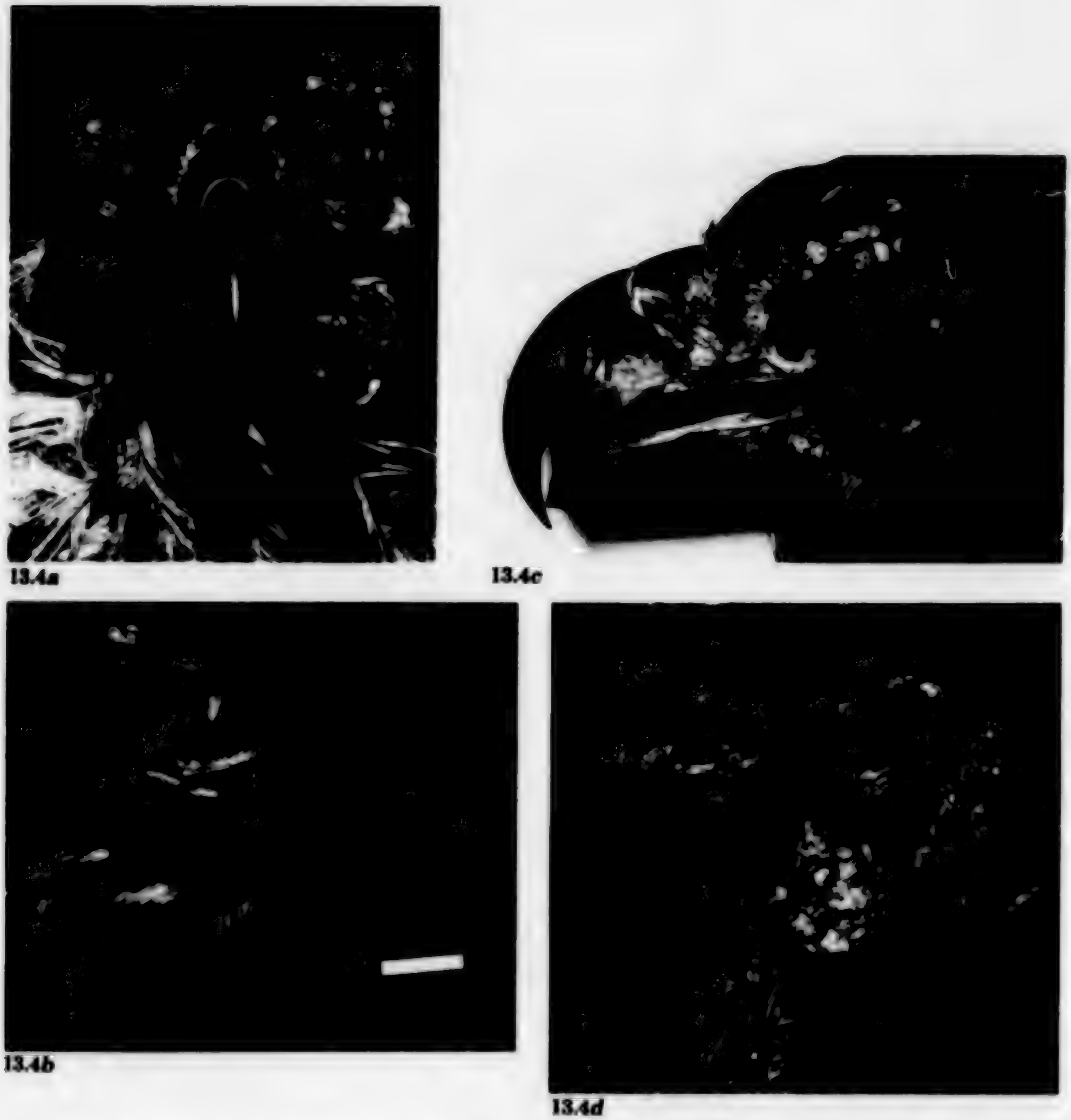
Fig. 13.3. (*a*) Avian pox lesions are typically found on unfeathered portions of the body. This Laysan albatross chick has small pox nodules on the face and eyelid. (*b*) As the disease progresses these lesions become more extensive. (*c*) Lesions are also commonly seen on the legs and (*d*) feet. (*a*) *W.H.*; (*b*, *c*, and *d*) *M.F.*

13.3c

13.3d

Gross Lesions

Avian pox can occur in two disease forms. The most common form consists of warty nodules that develop on the exposed surfaces of the bird. This form of the disease is usually self-limiting, with lesion regression and minor scarring occurring. However, these nodules can become enlarged and clustered, causing sight and breathing impairment and difficulty in feeding (Figs. 13.4*a* and *b*). Secondary tissue infections are common and contribute to bird mortality. The large warty nodules are subject to abrasions that become readily infected with bacteria and fungi (Figs. 13.4*c* and *d*). The less common diphtheritic form affects the mucous membranes of the upper digestive and respiratory tracts. This form of the disease has been reported only in a mute swan, a white-tailed tropicbird, a Laysan albatross, common murres, mourning doves, and northern bobwhite.



13.4a

13.4c

13.4b

13.4d

Fig 13.4. Pox lesions can be so extensive as to impair breathing, sight, and feeding as seen in these bald eagles. (*a*) Extensive involvement of both sides of the face , (*b*) obstruction to feeding due to the size and location of these lesions at the base of the bill, and (*c*) obstruction of sight due to complete occlusion of the eye. (*d*) Massive facial lesions often become abraded and subject to secondary infections. *J.R.*

Diagnosis

A presumptive diagnosis of avian pox can be made from the gross appearance of growths on body surfaces. However, these observations must be confirmed by examining lesions microscopically for characteristic inclusion bodies. Avian pox is confirmed by virus isolation and serological identification. Submit the whole bird or the affected body part (e.g., feet or head) to a diagnostic laboratory for examination. Place these samples in two plastic bags, refrigerate, and ship fresh in a leakproof container with wet ice or frozen chemical coolant packs as soon as possible after collection (see chapters on Specimen Collection and Preservation and Specimen Shipment). Immediately freeze samples that must be held for more than 24 hours before shipment. Virus isolation can be attempted from a living bird by collecting samples from the affected area. However, consult with the diagnostic laboratory staff before collecting samples.

Control

The fundamental principle for controlling poxvirus is to interrupt its transmission. The difficulty in applying control procedures is related to the type of transmission taking place, the mobility of the infected birds, and the size of the affected area. The more confined a population at risk, the more effective the control procedures will be. Vector control (primarily mosquitoes) in and around the disease area should be considered first. Identifying and eliminating vector breeding and resting sites together with control of adult mosquito populations are most desirable. Removing heavily infected animals is also helpful because it diminishes the source of virus to which the vector population can be exposed. This also reduces the opportunity for contact transmission between infected and noninfected birds.

Because poxvirus is resistant to drying, disease transmission by contaminated dust, food, perches, cages, and clothing can pose a continuing source of problems. Therefore, we highly recommend that these items be decontaminated with disinfectant, such as a 5% bleach solution, before disposal or reuse.

Modified live vaccines to prevent avian pox are used by the poultry industry, but their safety and effectiveness in wild birds have not been determined. In addition, the logistics of a vaccination program for wild birds would be prohibitive.

Human Health Considerations

Avian poxvirus is part of a larger family of poxviruses that includes the human disease known as variola or smallpox. However, the avipoxviruses have a limited host range among bird species, and there is no evidence that these viruses can infect man.

Supplementary Reading

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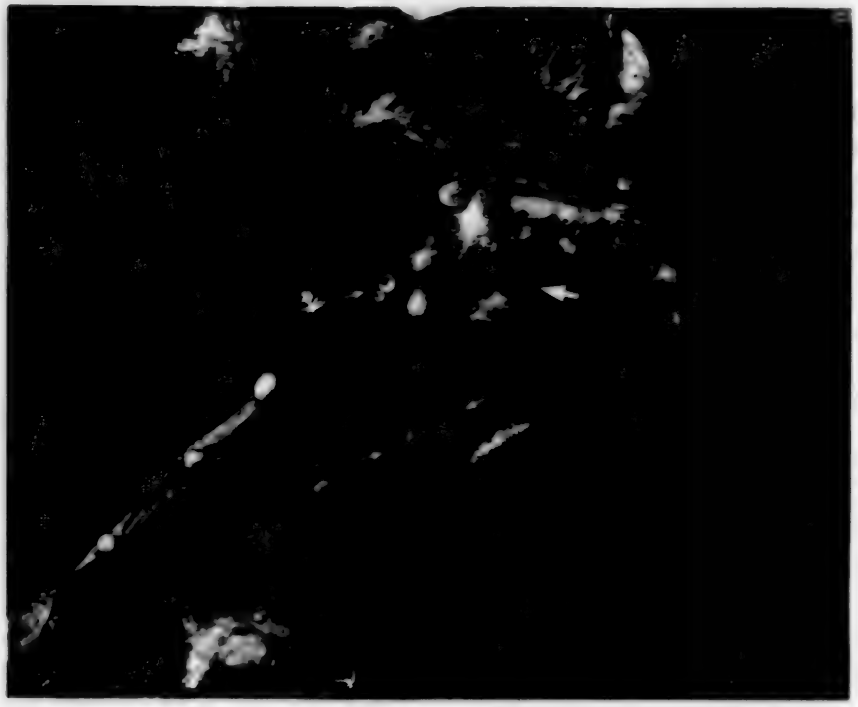
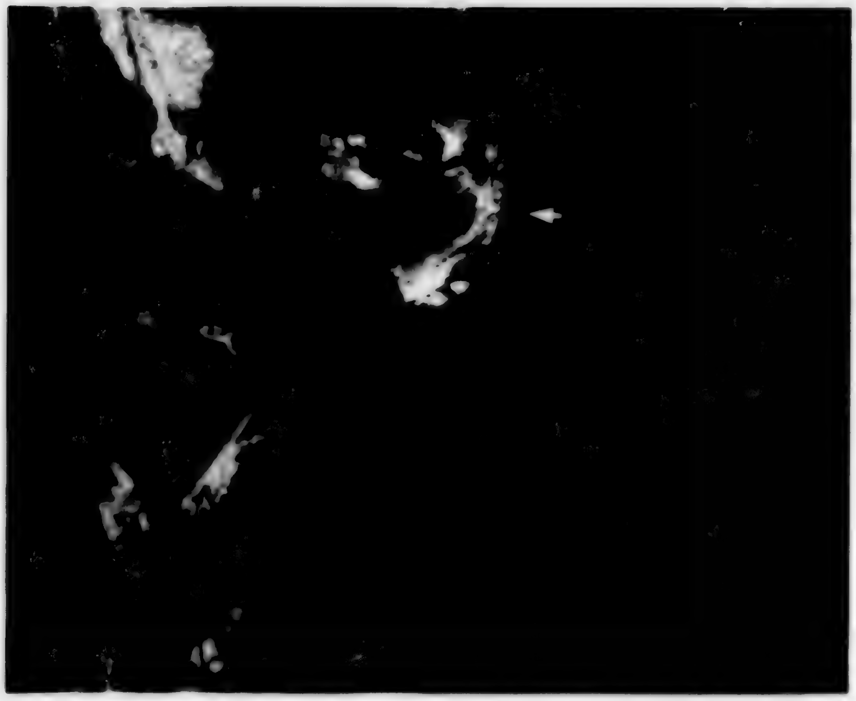
Fungal Diseases



14.4a



14.4b

Fig. 14.4. Lung of bird with chronic aspergillosis showing (*a*) "cheesy" fungal plaques, and (*b*) "bread mold" fungal mat totally involving the air sac adjacent to the heart of this bird. *M.F.*

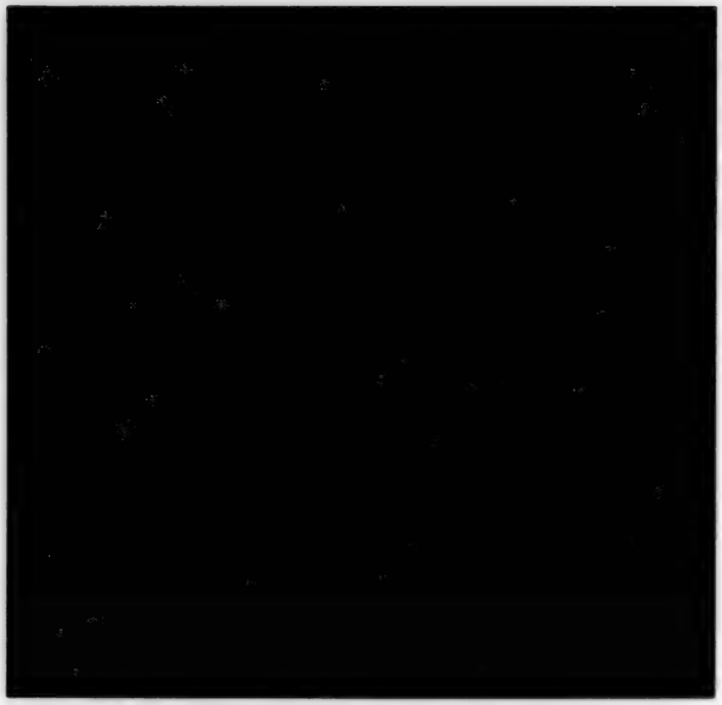


Fig. 14.5. Acute aspergillosis or "brooder pneumonia" in lung of a wood duck duckling. Note dark red, "studded" (granular) appearance of lung. *J.R.*

Human Health Considerations

Aspergillosis is not very contagious to humans. However, there are two sets of circumstances in which it can become a problem. (1) In individuals with severe, preexisting lung damage, such as widespread cancer in the lungs, aspergillosis may become a terminal secondary invader; and (2) a few individuals who have worked with *A. fumigatus* have become allergic to it and could develop an acute, life-threatening allergic reaction to this fungus. Other species of *Aspergillus* occasionally produce severe infections in birds and mammals, including dogs.

Supplementary Reading

Herman, C. M., and W. J. L. Sladen. 1958. Aspergillosis in waterfowl. Trans. N. Am. Wildl. Nat. Resour. Conf. 23:187–191.

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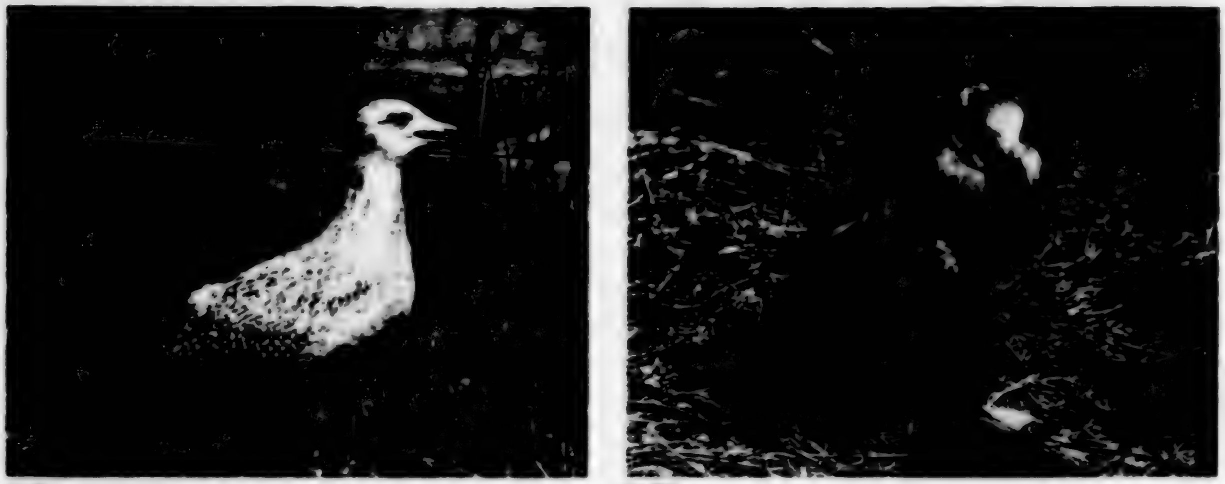

14.2a

14.2b

Fig. 14.2. (*a*) Respiratory distress and gaping (note the open bill) in a herring gull suffering from aspergillosis. (*b*) Wing droop also occurs. Note that the wing on the far side of this bird is drooping well below the body. *M.F.*

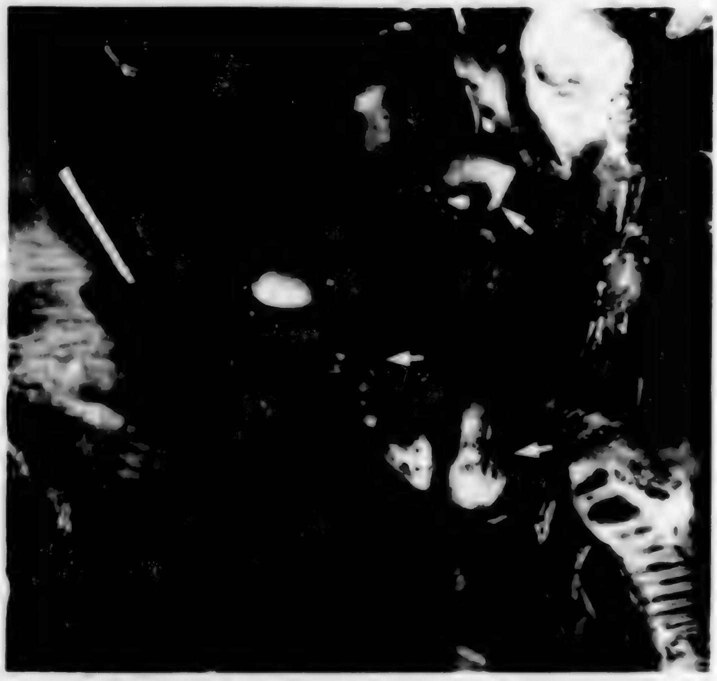


Fig. 14.3. "Cheesy" plaques in the lungs and air sacs of a bird with aspergillosis. *M.F.*

Field Signs

The typical aspergillosis-affected bird is emaciated and frequently manifests severe and progressive difficulty in breathing by gaping (rapid opening and closing of the bill; Fig. 14.2*a*). Birds often appear to be unthrifty, and wing-droop may occur (Fig. 14.2*b*). Infected birds are usually weak and may fail to try to escape. With the exception of visible evidence of breathing difficulties, these signs are similar to those for lead poisoning. Infection that reaches the brain can result in obvious loss of muscular coordination and twisting of the head and neck so that the head is held in unnatural positions.

Epizootic aspergillosis and "brooder pneumonia" outbreaks are often characterized by sudden deaths of previously healthy birds. Sick birds show acute respiratory distress and failure.

Gross Lesions

Birds infected with the more typical "chronic" form of aspergillosis usually have variously sized lesions in the lungs and air sacs. Typically, these lesions appear as flattened yellow plaques with a "cheesy" appearance and consistency (Fig. 14.3). Aggregations of these lesions may completely line the air sac as a continuous mass. There may also be an extensive growth of the fungus on tissue and plaque surfaces that appears similar to bread mold. This velvety blue-green or grey fungal mat is striking in appearance (Fig. 14.4).

In cases of acute aspergillosis, the birds are usually in good flesh, with good-to-moderate deposits of fat. Air sacs are usually thickened, but the most striking lesion is a dark red, firm lung that is often studded ("peppered") with small, 1–2 mm yellow nodules (Fig. 14.5).

Diagnosis

Diagnosis is based on finding the typical lesions at necropsy and on isolating the fungus from the tissues. *Aspergillus* spp. can be identified by microscopically examining material from fungal mats seen at necropsy and from tissue sections that have been specially stained. However, the specific species of *Aspergillus* cannot be identified by these means.

Control

The spores of the mold *A. fumigatus* are widely distributed and are often present in moldy feeds, unclean brooders and incubators, moldy straw, and rotting agricultural waste. *Aspergillus* grows best on decomposing organic matter left in a warm, dark, moist environment. Failure to maintain a clean environment often leads to severe outbreaks.

Outbreaks of aspergillosis have occurred in mallards feeding in fields previously covered by discarded moldy corn silage. Dusty straw hay placed as litter in the bottoms of wood duck nest boxes has resulted in losses of wood duck ducklings. Avoid using moldy or dusty straw, silage, or feed, and dumping moldy waste grain in areas where waterfowl and other birds feed. Birds should be denied the use of fields where moldy agricultural waste products such as waste corn, peanuts, straw, or hay have accumulated.

Fig. 14.1. Relative occurrence of aspergillosis in wild birds in North America.

Type of bird	Frequency of occurrence
Waterfowl	● ● ●
Herons	●
Shorebirds	○
Gulls	● ● ●
Raptors (free-living/captive)	●/ ● ● ●
Upland game birds	● ●
Crows, ravens	● ● ●
Blackbirds, cowbirds, grackles	● ●
Songbirds	● ●

● ● ●	Frequent
● ●	Occasional
●	Infrequent
○	Rare or not reported

Chapter 14.
Aspergillosis

Louis N. Locke

Synonyms

"Asper," brooder pneumonia
(in chickens and ducklings)

Cause

Aspergillosis is an infection of the respiratory tracts of birds and mammals caused by fungi of the genus *Aspergillus*. *Aspergillus fumigatus* is the species most frequently involved. Although the disease is caused by an infectious agent, it is not contagious (i.e., it will not spread from bird to bird). Infection usually results from the inhalation of *Aspergillus* spores.

Species Affected

A wide variety of birds have died of aspergillosis and probably all birds are susceptible. Aspergillosis was one of the first bird diseases described, having been noted in a scaup in 1813 and in a European jay in 1815. It is frequently seen in waterfowl and loons, and is a major problem in captive raptors (Fig. 14.1). Young birds seem to be much more susceptible than adults. Aspergillosis is commonly diagnosed as a cause of death in captive penguins.

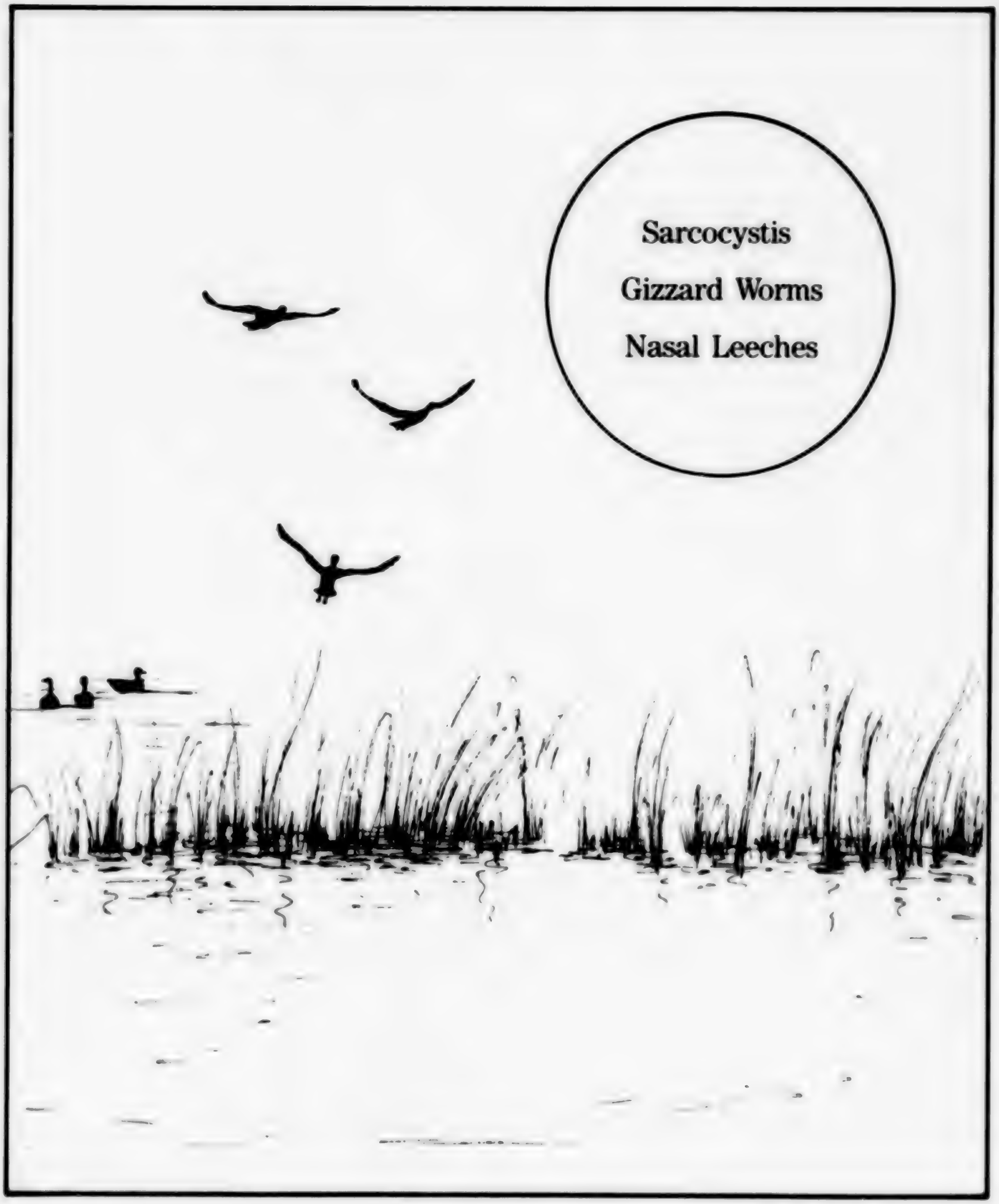
Distribution

Aspergillosis has a nearly worldwide reported occurrence in animals, the Antarctic being a notable exception. Although major bird die-offs have been reported within the United States and elsewhere, including Colorado (mallards), South Dakota (mallards), Massachusetts (Canada geese), Missouri (Canada geese), Illinois (wood ducks, ducks), California (mallards), Ontario, Canada (American black ducks), and New Zealand (black swans), aspergillosis usually occurs as scattered, individual cases.

Seasonality

Most aspergillosis outbreaks in waterfowl occur in fall to early winter; individual cases can occur at any time, particularly among birds stressed by crippling, oiling, malnutrition, recent capture, and concurrent disease conditions. Most cases are seen after hunting season. This disease is a frequent complication in hunter-crippled waterfowl, among birds on nutritionally deficient diets, and in birds whose immune systems have been compromised by exposure to environmental contaminants such as lead. Aspergillosis can cause serious losses among seabirds in rehabilitation programs associated with oil spills. Environmental factors also contribute to the time of year when aspergillosis is seen. Scattered outbreaks of this disease occurred among American coots, diving ducks, tundra swans, and passerine birds throughout California one winter at the end of a 3-year drought. Severe dust conditions associated with this weather pattern are thought to have interfered with respiratory clearance mechanisms, thereby increasing bird susceptibility to this disease. Brooder pneumonia, a specialized springtime form of aspergillosis, occurs when chicks or ducklings are placed in *Aspergillus*-contaminated brooders. Catastrophic losses have occurred on game farms under these circumstances. Aspergillosis is also an important cause of mortality in winter roosts of blackbirds in Maryland and Pennsylvania.

Parasitic Diseases



Sarcocystis

Gizzard Worms

Nasal Leeches

Chapter 15.
Sarcocystis

Benjamin N. Tuggle

Synonyms

Rice breast disease, sarcosporidiosis, sarcocystosis

Cause

Sarcocystis is a nonfatal, usually asymptomatic parasitic infection caused by a protozoan. Various species of this parasite affect mammals, reptiles, and birds. The most commonly reported species in North American waterfowl is *Sarcocystis rileyi*. Infection occurs when waterfowl ingest food or water that contains eggs of the parasite.

Species Affected

Dabbling ducks (mallard, northern pintail, northern shoveler, teal, American black duck, gadwall, and American wigeon) commonly have visible (macroscopic) forms of *Sarcocystis* spp.; these forms are far less frequently found in other species of ducks and infrequently found in geese and swans. Microscopic forms of infection are frequently found in snow geese, Canada geese, and a variety of duck species. Land birds such as grackles and other passerine bird as well as mammals and reptiles, also can have visible forms of sarcocystis, but it is unlikely that *S. rileyi* is the species of parasite involved. With the exception of waterfowl, this parasite has received little study in migratory birds. This must be taken into account when considering current knowledge of species affected (Fig. 15.1).

Distribution

Sarcocystis is a common parasitic infection of some species of waterfowl and occurs throughout the geographic range of those species in North America. Sarcocystis infections of waterfowl have also been reported from Australia and other countries.

Seasonality

Infected birds can be found year-round, but waterfowl infected with *Sarcocystis* spp. are usually observed during the hunting season. Infection is not seen in prefledgling waterfowl, nor is it often seen in juveniles. Two possible reasons for these differences between age-classes may be that the development of visible forms of the parasite requires time, or infection may not occur until after the birds leave their breeding grounds. Because visible forms of sarcocystis are more frequently developed in older birds, hunter detection tends to be greatest during years of poor waterfowl production when the bag contains a greater proportion of adult birds. Too little is known about sarcocystis in other groups of migratory birds to evaluate seasonality.

Field Signs

Usually there is no externally visible sign of this disease, nor is this disease recognized as a direct cause of mortality in migratory birds. Heavy infections can cause loss of muscle tissue and result in lameness, weakness, and even paralysis in rare instances. The debilitating effects of severe infections could increase susceptibility to predation and other causes of mortality.

Gross Lesions

Visible forms of infection are readily apparent once the skin is removed from the bird. The cream-colored, cylindrical cysts resemble grains of rice running in parallel streaks through the muscle tissue. They commonly occur throughout the breast muscle (Fig. 15.2*a*), but can also occur in other skeletal and cardiac muscle (Fig. 15.2*b*). Calcification of the muscle tissue around these cysts makes them obviously discrete bodies. The degree of calcification is often sufficient to give a gritty feeling to the tissue when it is cut with a knife.

Diagnosis

The visible presence of sarcosporidian cysts in muscle tissue is sufficient to diagnose this disease. Visible cysts may vary in size and shape in different bird species. Good quality color photographs (prints or 35mm slides) showing the external surface of infected muscle are generally sufficient for a disease specialist to recognize this disease if tissues or the whole carcass cannot be provided. Whole birds should be submitted if possible. If only tissues can be submitted, a portion of the infected muscle tissue should be fixed in a 10% formalin solution. Frozen muscle tissue is also suitable for diagnosis, and the distinctive appearance of these cysts allows a diagnosis in even partially decomposed carcasses.

Control

There are no known control methods for this disease, nor do any seem to be needed or are any being developed. Control of sarcocystis would require interruption of the life cycle of the parasite. Although the life cycles of the *Sarcocystis* species affecting waterfowl are not precisely known, they are probably similar to the two-host life cycle known for some other species of *Sarcosystis* (Fig. 15.3). The predator–prey relation between the intermediate hosts (waterfowl) and the definitive hosts (carnivores) may be the primary reason that juvenile waterfowl or some species of waterfowl are seldom found to be infected. The appropriate carnivores may not be present on the breeding grounds.

Waterfowl become infected by ingesting the eggs (oocysts) of the parasite in food or water, the contamination originating from carnivore feces. Development of the parasite continues within the intestine of the bird where an intermediate form (sporozoites) enters the bloodstream and infects specific cells of the blood vessels. Multiplication of these cells gives rise to a second intermediate form (merozoites) that is carried by the blood to voluntary muscles where elongated cysts are eventually produced. The life cycle is completed when a carnivore ingests the infected muscle tissue of a bird (Fig. 15.3). Infection in the carnivore occurs only in the intestine, does not involve macrocyst formation, and rarely causes illness or other forms of disease.

The life cycle is further complicated by the knowledge that different species of carnivores seem to be involved in infection of different waterfowl species. From a parasitologist's viewpoint, this suggests that waterfowl are infected by more than one species of the genera *Sarcocystis*. If the carnivore–waterfowl cycle is species-specific (that is, a specific species of waterfowl can only be infected by oocysts from a specific carnivore species), selective control of *Sarcocystis* might be feasible. However, current knowledge of the disease does not indicate a need to initiate control. This information is of potentially greater value for the maintenance of captive waterfowl flocks. Also, the role of carnivores in the life cycle of *Sarcocystis* infections needs to be considered when feeding uncooked infected waterfowl to house pets and farm animals such as hogs.

Human Health Considerations

Sarcocystis presents no known health hazard to humans. The primary importance to humans of sarcocystis in wild waterfowl is loss of infected birds for food; the unaesthetic appearance of parasitized muscle generally prompts hunters to discard the carcass. Also, the recognized high prevalence of infection in northern shovelers in some areas results in this species often being left unretrieved by some hunters and focuses additional hunting pressure on other species.

Fig. 15.1. Relative frequency of grossly visible forms of sarcocystis in selected groups of North American migratory birds.

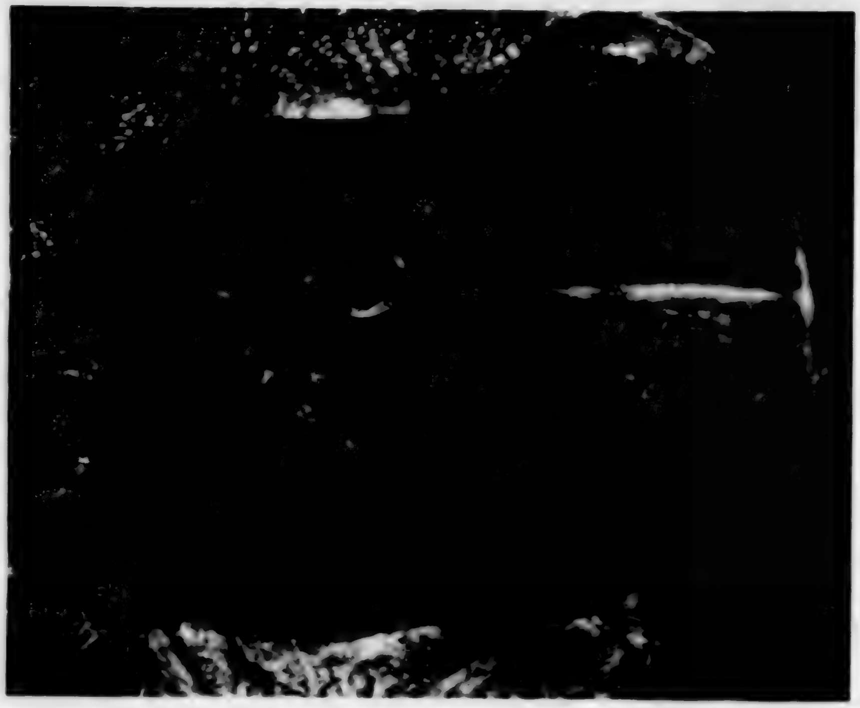
Type of bird	Expected occurrence
Puddle ducks	● ● ●
Diving ducks	●
Mergansers	●
Sea ducks	●
Geese	●
Swans	●
Wading birds (herons, egrets, ibis)	○

● ● ● Frequent
● ● Often
● Occasional
○ Rare or not reported

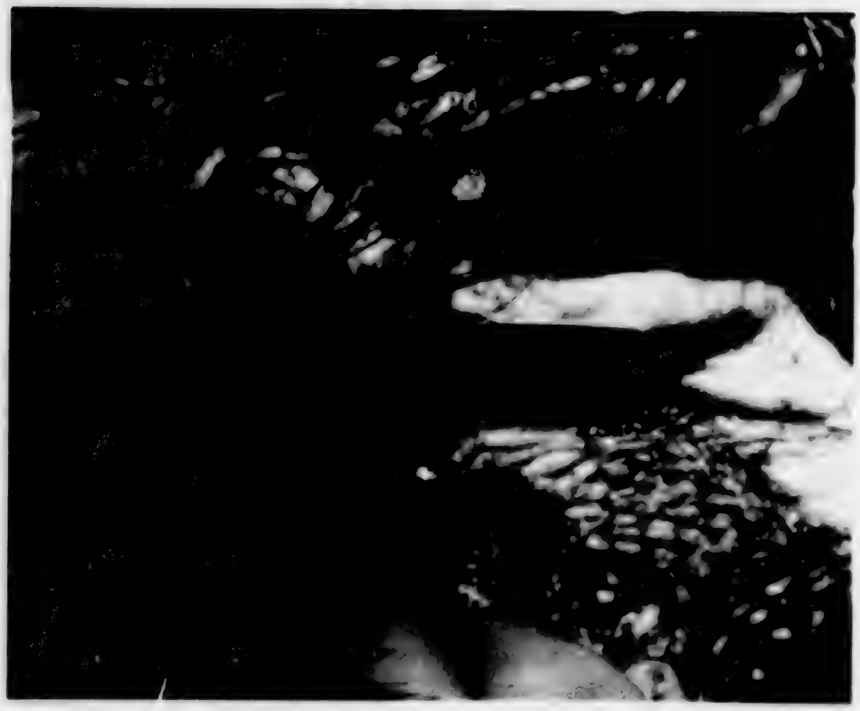
Fig. 15.1. Relative frequency of grossly visible forms of sarcocystis in selected groups of North American migratory birds. (Continued)

Type of bird		Expected occurrence
Shorebirds		○
Gulls and terns		○
Pelicans		○
Raptors		○

● ● ● Frequent
● ● Often
● Occasional
○ Rare or not reported



15.2*a*



15.2*b*

Fig. 15.2. Rice-grain-sized cysts of *Sarcocystis* spp. evident in parallel streaks (*a*) in breast muscle fibers of a mallard and (*b*) in thigh and leg muscle of an American black duck. *J.R.*

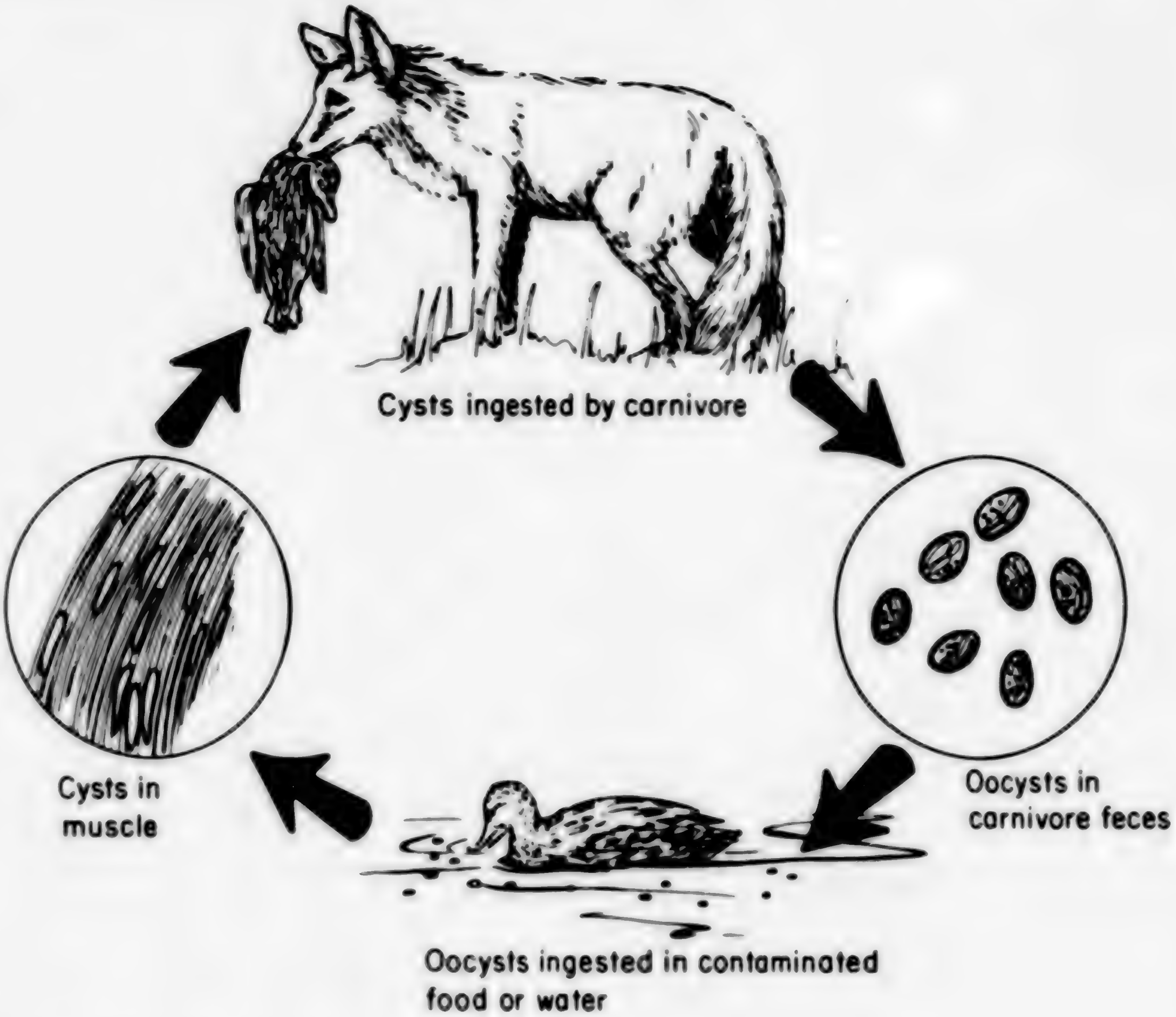

Cysts ingested by carnivore
Oocysts in carnivore feces
Oocysts ingested in contaminated food or water
Cysts in muscle

Fig. 15.3. General life cycle of *Sarcocystis* spp.

Supplementary Reading

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Chapter 16.
Gizzard Worms

Benjamin N. Tuggle

Synonyms

Stomach worm, ventricular nematodiasis

Cause

Several species of parasitic nematodes in migratory birds are referred to as gizzard worms. The two discussed here are trichostrongylid nematodes belonging to the genera *Amidostomum* and *Epomidiostomum*. These long (10–35 mm), sometimes coiled, threadlike roundworms are located just beneath the surface lining and the grinding pads of the gizzard and are most frequently found in waterfowl. Infestation results from ingestion of infective gizzard worm larvae. Severe infestations can result in birds being debilitated to the extent that they become more susceptible to predation and infection from other disease agents.

Species Affected

Gizzard worms can be found in a variety of migratory birds and have been reported in ducks, geese, swans, American coot, and grebes (Fig. 16.1). Mortality can occur and is more common in very young birds than in adults. These worms are more prevalent in geese than in ducks or swans. Infestation is most severe in snow and Canada geese.

Distribution

Gizzard worms have a worldwide distribution and were first reported in domestic geese from Europe.

Seasonality

Migratory birds first become exposed to gizzard worms on the breeding grounds and can continue to be exposed throughout their lives. Therefore, no seasonality is associated with this parasitism. The loss of young birds may be particularly high during fall and winter months because of the combined effects of large worm burdens, stresses of migration, and competition for food.

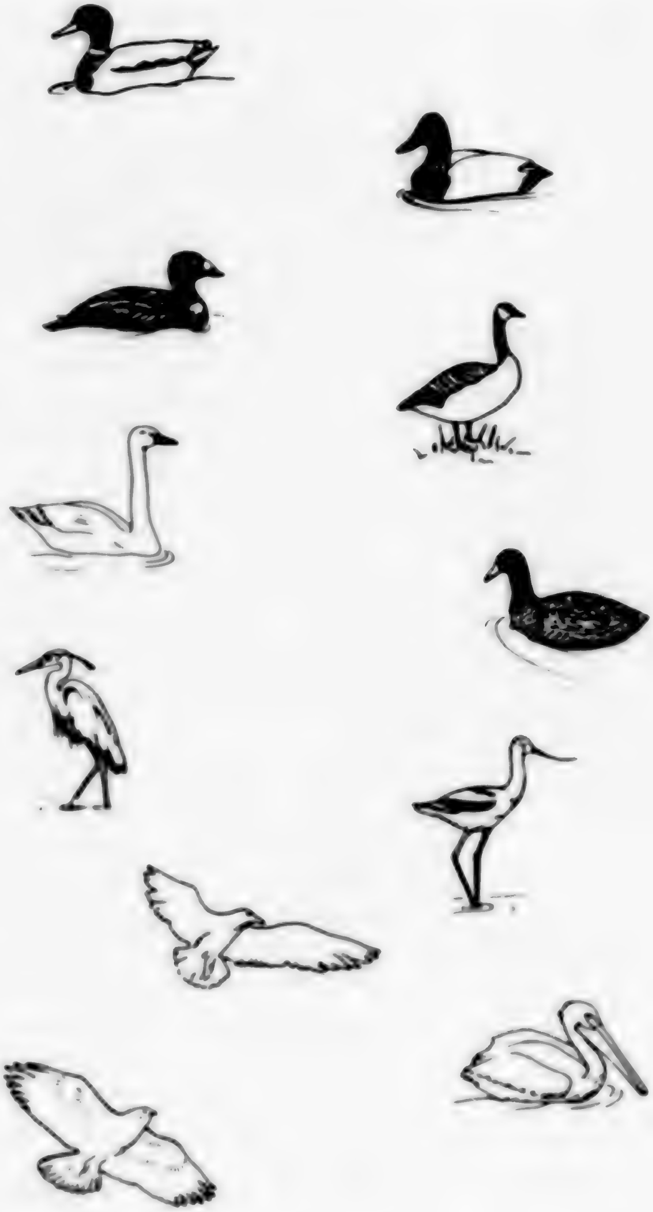
Field Signs

There are no field signs indicative of gizzard worm infestation. Heavy worm burdens can result in poor growth of young birds, and birds of all ages are subject to emaciation and general weakness. Severe infections can interfere with food digestion by the bird as a result of extensive damage to the gizzard lining and muscle.

Gross Lesions

Obvious changes from the normal appearance of the gizzard result from the development, migration, and feeding of gizzard worms in that organ. Sloughing, inflammation, hemorrhages, and ulceration of the gizzard lining can occur with erosion of the grinding pads (Fig. 16.2). Large numbers of worms (> 35) can denude the surface lining of the gizzard, causing the edges of the grinding pads to degenerate and separate the pads from the underlying tissue (Figs. 16.3*a,b,c,* and *d*). In geese, portions of the gizzard muscle can become necrotic due to the presence of variable numbers of *Epomidiostomum* spp., which migrate through the tissue. Oblong tissue cavities 1–4 cm long can also be present (Fig. 16.4) and contain granular material resulting from tissue reaction to worm migration through the muscle.

Fig. 16.1. Relative frequency of trichostrongylid nematodes in the gizzards of selected groups of North American migratory birds.



Type of bird	Expected occurrence
Puddle ducks	●
Diving ducks	●
Sea ducks	●
Geese	● ● ●
Swans	● ●
Coots	● ●
Wading birds	○
Shorebirds	○
Gulls and terns	○
Pelicans	○
Raptors	○

● ● ● Frequent
● ● Often
● Occasional
○ Rare or not reported

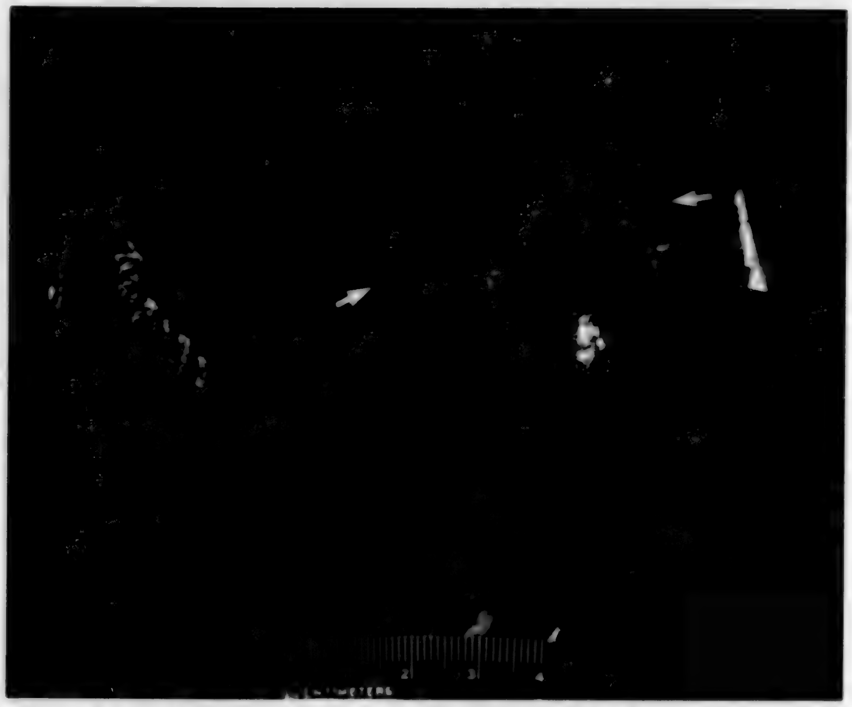


Fig. 16.2. Canada goose gizzard showing ulcerations in gizzard lining caused by gizzard worm (*Amidostomum* spp.) infection. *J.R.*

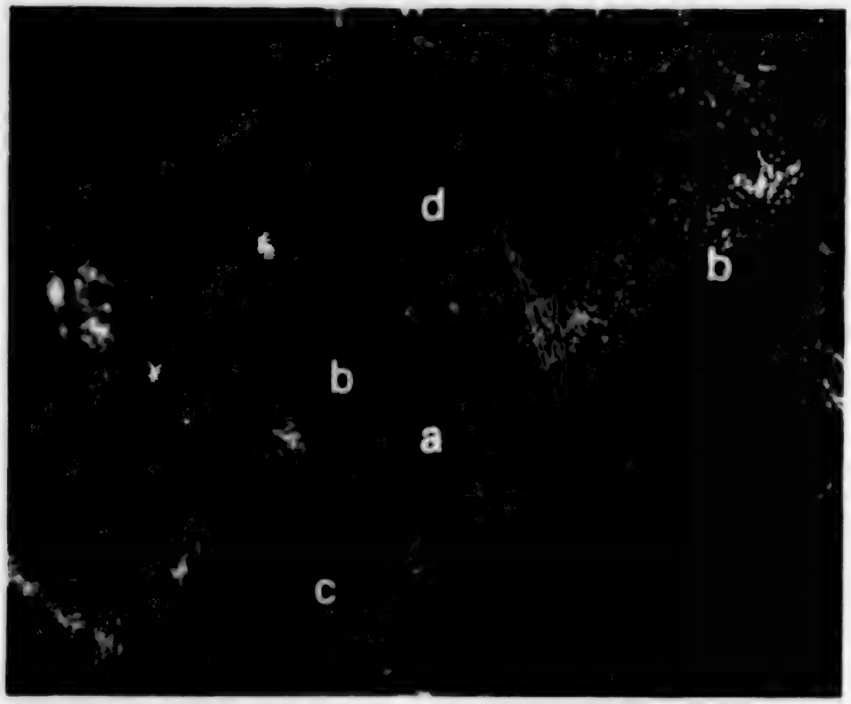


Fig. 16.3. Closeup shot of Canada goose gizzard showing (*a*) denuded surface lining and (*b*) degeneration of the edges of the grinding pads. Note also (*c*) separation of pads from gizzard lining and (*d*) presence of worms. *J.R.*

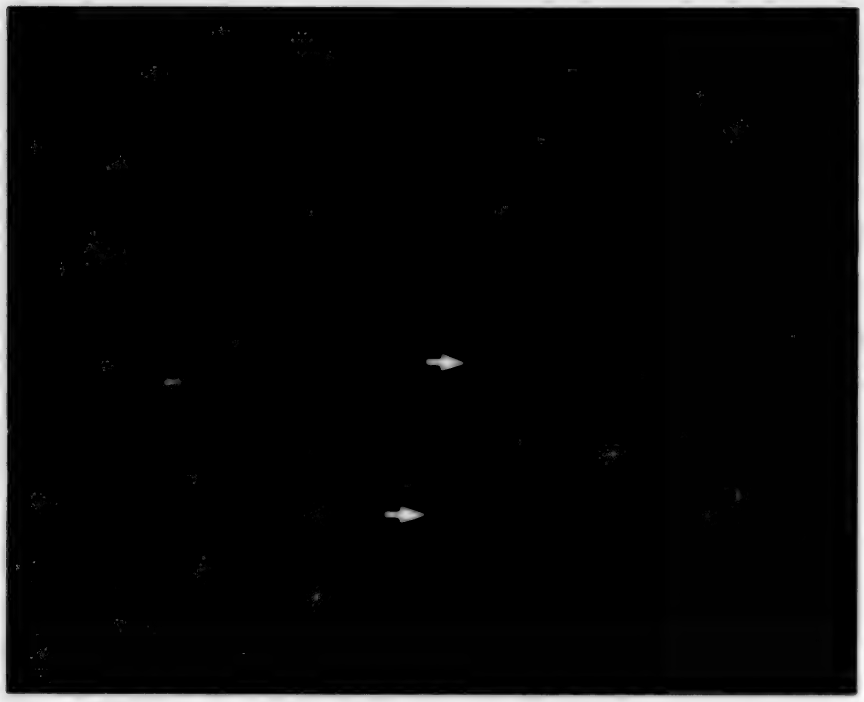


Fig. 16.4. Areas of tissue destruction and reaction to migrating *Epomidiostomum* spp. in the gizzard muscle of a snow goose. *J.R.*

Diagnosis

Gizzard worm infestation can be determined in live birds by finding and identifying gizzard worm eggs in the feces. The eggs of *Amidostomum* spp. and *Epomidiostomum* spp. are similar in size and appearance and require speciation by trained individuals.

Large numbers of worms and lesions in the gizzard lining or muscle of carcasses are highly suggestive of death caused by gizzard worms. Submit carcasses to disease diagnostic laboratories for more thorough evaluations. If this is not possible and you suspect gizzard worms as the cause of mortality, remove the gizzard and ship chilled or frozen. If the gizzard has been opened, remove as many whole worms as possible with forceps and place them in a 10% formalin or a 70% ethanol solution. These worms should not be frozen. Submit the opened gizzard with the worms, or preserve slices of the gizzard muscle in 10% formalin and forward them for microscopic examination (see chapter on Collection and Preservation of Specimens).

Control

Methods for controlling gizzard worms in free-living migratory birds have not been developed. Attempts to do so would involve disruption of the parasite's life cycle. Gizzard worms have a direct life cycle (Fig. 16.5). Embryonated eggs are passed in the feces of the bird. First-stage larvae hatch from eggs into the surrounding environment in about 24–72 hours, depending on temperature. These larvae undergo two molts after hatching, with time between molts being temperature dependent. Larvae are quite resilient, surviving low temperatures and even freezing. However, they do not survive drying.

After ingestion by the host the larvae enter the gizzard and burrow into the surface lining where they molt again before becoming adult worms. Infective larvae are most commonly ingested while the bird feeds or drinks. Adult worms become sexually mature in about 10–15 days after the final molt and females shed eggs within 15–20 days. The development from egg to adulthood may take as few

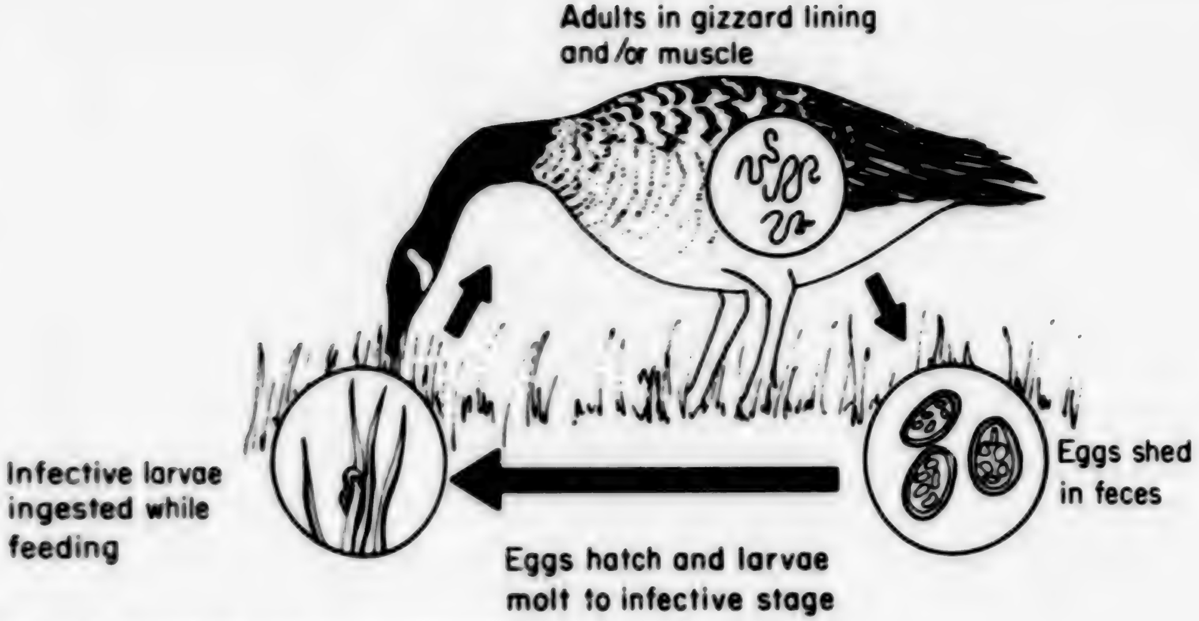

Adults in gizzard lining and/or muscle
Eggs shed in feces
Eggs hatch and larvae molt to infective stage
Infective larvae ingested while feeding

Fig. 16.5. General life cycle of trichostrongylid gizzard nematodes.

as 20 days or as many as 35 days depending on environmental conditions. Once infected, a bird can harbor gizzard worms for several years.

This direct life cycle suggests that transmission potential is greatest in crowded and continuously used habitat because of accumulative fecal contamination, provided that ambient temperatures are warm enough for larval development (20–25 °C). Newly hatched birds are least resistant to infestation, and birds of all ages are susceptible to reinfestation.

Human Health Considerations

Gizzard worms are not a threat to man. People who eat gizzards of waterfowl should cook them thoroughly and discard those that appear unhealthy.

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Chapter 17.
Nasal Leeches

Benjamin N. Tuggle

Synonym

Duck leeches

Cause

Bloodsucking leeches of the genus *Theromyzon* are the only leeches in North America known to feed directly in the nasal passages, trachea, and beneath the nictitating membrane of the eyes of migratory birds. Three species of nasal leeches have been reported from North America, *T. rude*, *T. tessulatum*, and *T. biannulatum*. There are other genera of leeches that feed on the exposed surfaces of waterfowl.

Species Affected

Nasal leeches affect many aquatic bird species (Fig. 17.1). Affected waterfowl include northern pintail, teal, American wigeon, northern shoveler, ring-necked duck, canvasback, redhead, lesser scaup, bufflehead, gadwall, ruddy duck, white-winged scoter, surf scoter, trumpeter swan, and tundra swan. Geese may also be parasitized but less frequently than ducks and swans.

Distribution

Nasal leech infestations of waterfowl and other migratory birds have not been reported south of the 30th parallel and are most commonly observed in northern areas because these parasites are better adapted to cold-water lakes (Fig. 17.2).

Seasonality

Peak parasitism usually occurs in the spring and summer months. During this time leeches are actively seeking potential hosts and reproducing. During the winter months the ambient temperatures in frozen ponds and marshes considerably slow their metabolic rate and thus their activity. In wetlands kept free of ice during the winter, bird activity may stimulate opportunistic feeding by leeches.

Field Signs

Birds that have leeches protruding from the nares or attached externally to the mucous membranes of the eyes are easily recognized from a distance with the aid of binoculars (Fig. 17.3). Leeches may be so blood-engorged that they resemble small sacks of blood (Fig. 17.4). Infested birds may be seen vigorously shaking their heads, scratching at their bills with their feet, or sneezing in an effort to dislodge the leeches and to force air through blocked nasal passages. These efforts are usually unsuccessful. Nasal and respiratory tract leech infestations can cause labored breathing and gaping similar to that seen among birds suffering from aspergillosis.

Gross Lesions

Severe leech infestations of the eye can result in temporary blindness. Eye damage may be seen as an accumulation of a stringy, cheeselike material beneath the nictitating membrane, as clouding of the cornea, and in some instances as collapse of the globe of the eye. Nasal passages (Fig. 17.5), throat, and trachea can become blocked by engorged leeches. The feeding action of *Theromyzon* spp. can cause inflammation and extensive damage to the lining of the nasal cavity.

Fig. 17.1. Relative frequency of nasal leech infestations in selected groups of migratory birds.

Type of bird		Relative frequency
Puddle ducks		● ● ●
Diving ducks		● ●
Mergansers		●
Sea ducks		●
Geese		●
Swans		● ● ●
Grebes and loons		●

● ● ● Frequent
● ● Often
● Occasional
○ Rare or not reported

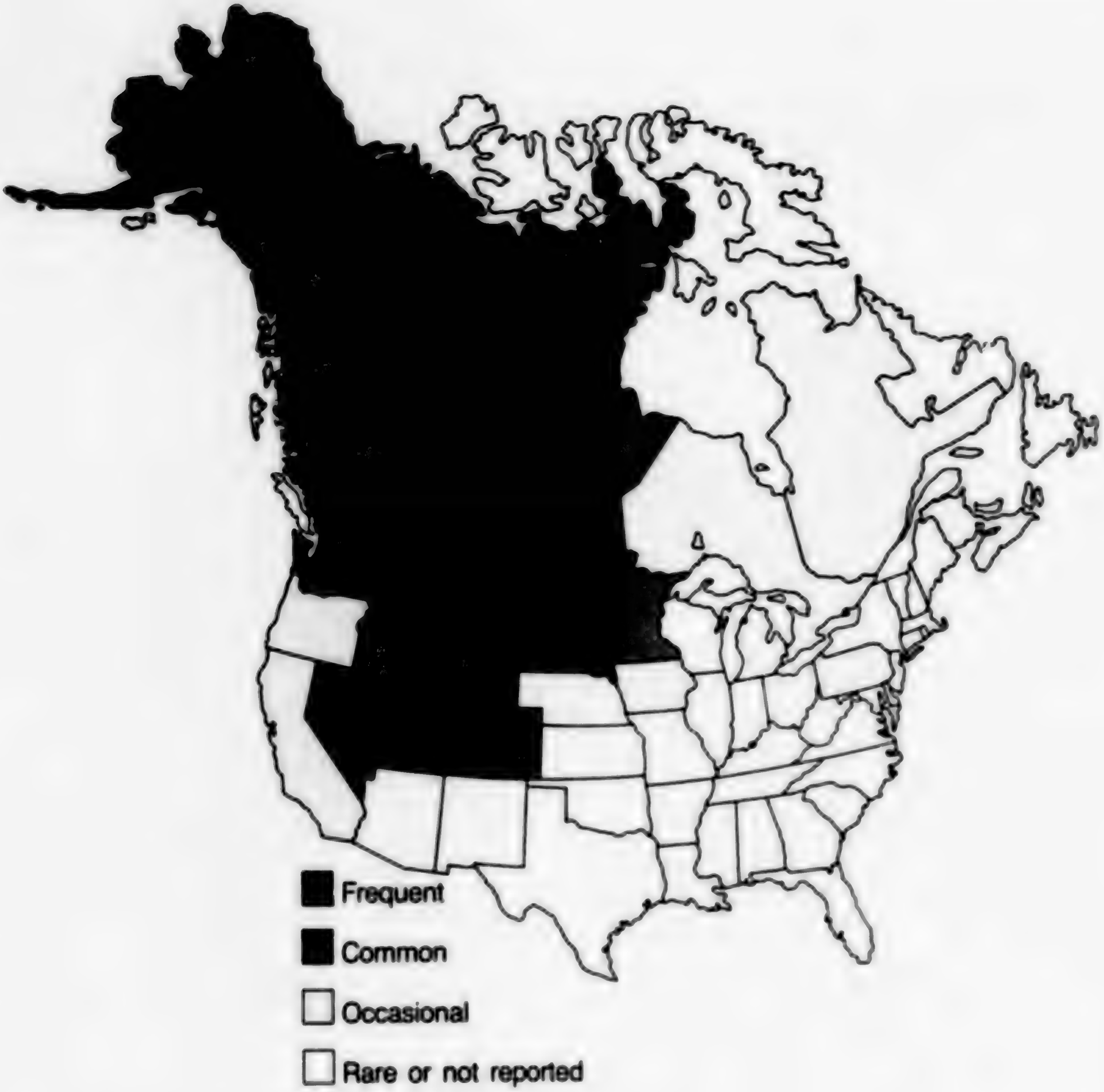


Fig. 17.2. Reported distribution of *Theromyzon* spp. in North America.

17.3a

17.3b

Fig. 17.3. External nasal passage infestations of nasal leeches on (a) eyes and (b) nares. *DT*

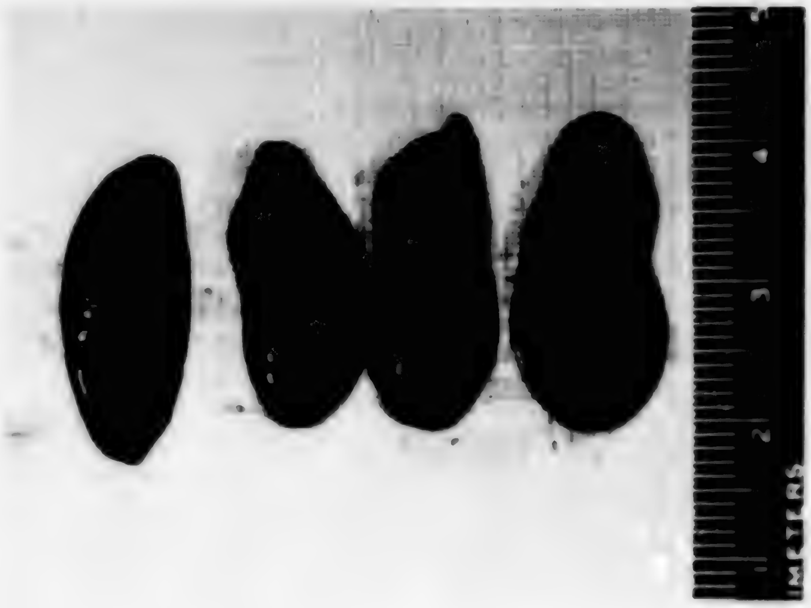


Fig. 17.4. Blood-engorged *Theromyzon* sp. removed from the nares of a trumpeter swan. *J.R.*

Diagnosis

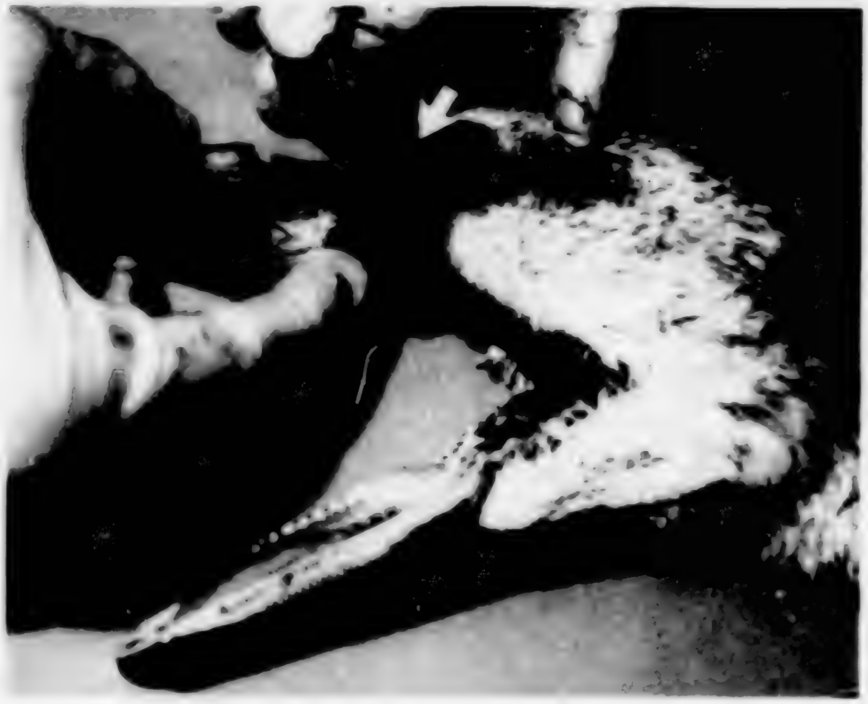
Nasal leeches are 10–45mm long when blood-engorged, amber or olive colored, and have four pairs of eyes. Those found in the free-living state are green, with variable patterns of spots on the top surface. Diagnosis of parasitism is usually made by seeing blood-engorged leeches protruding from the nares or attached to the eyes (Fig. 17.3), especially in birds that cannot be handled. Birds with internal leech infestations cannot be diagnosed by observation. In cases where the suspected cause of death is nasal leech parasitism, submit the entire carcass for examination. Leeches may depart a dead bird, making diagnosis difficult, or may move to other areas of the body where they may be overlooked. Therefore, leeches found on carcasses should be collected and submitted with the carcass. They can be shipped alive in pond water and can be maintained in that condition for several months if kept refrigerated.

If leeches are to be killed before shipment, it is necessary to preserve them in a relaxed state so that species identification can be made. Straighten specimens between two glass slides (Fig 17.6*a*) and flood with a 10% formalin solution for 3 to 12 minutes while applying pressure to the top microscope slide (Fig. 17.6*b*); then place them in a 10% formalin solution for about 12 hours to complete the fixation. Afterwards, transfer leeches to a 5% formalin solution for preservation (Fig. 17.6*c*).

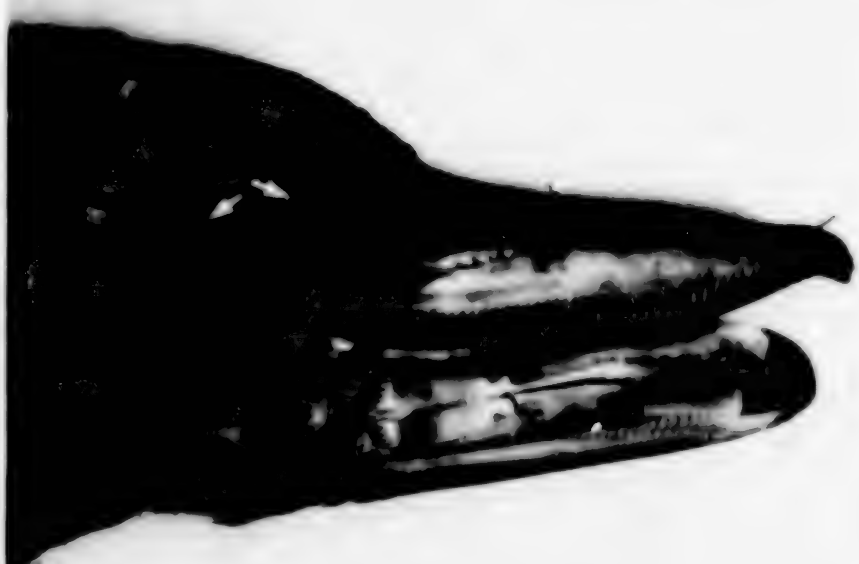
Control

Leech infestations in waterfowl are common and can be fatal, especially in cygnets and ducklings. Tissue damage resulting from heavy infestations can faciliate secondary bacterial infections. However, no preventive measures have been developed for combating infestations in wild birds. When not feeding on birds, nasal leeches occur as free-living organisms in aquatic environments. Control measures to reduce leech populations might be possible if nasal leeches could be selectively killed. However, leech control must be weighed against the value of leeches as an aquatic bird food item.

Leeches protruding externally from the nares or attached to eyes can be removed with forceps. Those present in the nasal passages can be removed by submerging the bird's bill for 5 to 10 seconds in a 10–20% salt solution. This can be repeated several times if necessary.



17.5a



17.5b

Fig. 17.5. (*a*) Nasal passages of trumpeter swan infested with blood-engorged *Theromyzon* leeches. (*b*) Dissected nasal passages of a mallard showing leech infestation in sinus passages. *J.R.*

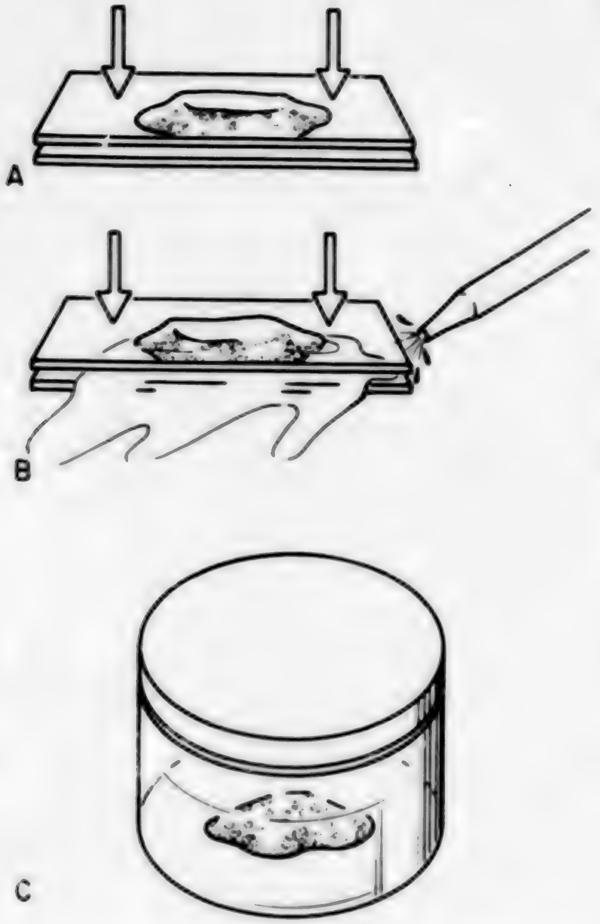


Fig. 17.6. Leech fixation procedure. (*a*) Straighten (flatten) leech between two glass slides; (*b*) flood area between slides with 10% formalin for 3–5 minutes while maintaining downward pressure on top slide; and (*c*) transfer leech to container of 10% formalin (see text for additional details).

Human Health Considerations

Theromyzon spp. feed exclusively on avian hosts and are not considered a threat to humans.

Supplementary Reading

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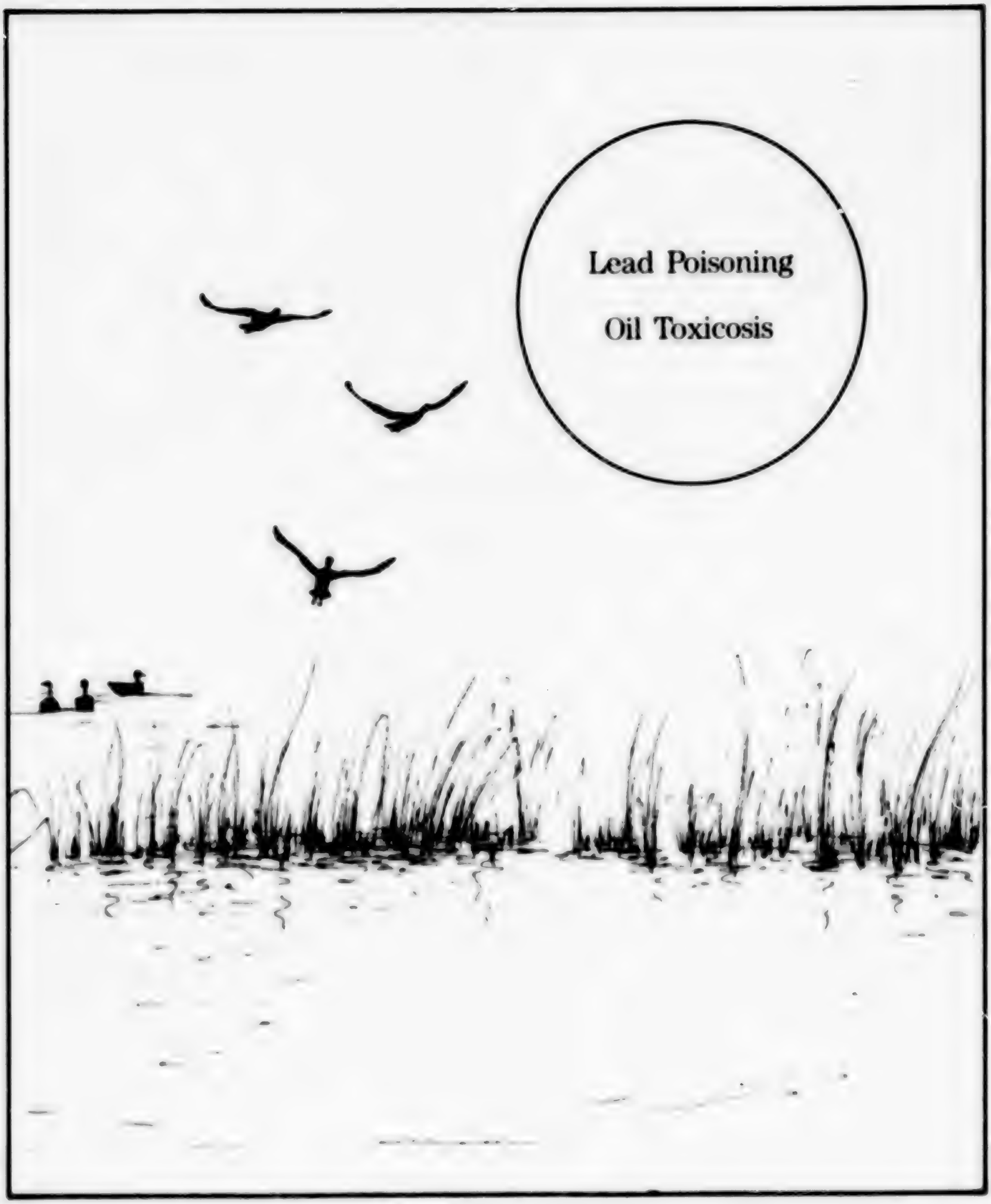
Toxic Diseases



Lead Poisoning

Oil Toxicosis

Chapter 18.
Lead Poisoning

Milton Friend

Synonym

Plumbism

Cause

Lead poisoning is an intoxication resulting from absorption of hazardous levels of lead into body tissues. Lead pellets from shot shells, when ingested, are the most common source of lead poisoning in migratory birds. Other far less common sources include lead fishing sinkers, mine wastes, paint pigments, bullets, and other lead objects that are swallowed.

Species Affected

Lead poisoning has affected every major species of waterfowl in North America and has also been reported in a wide variety of other birds. The annual magnitude of lead poisoning losses for individual species cannot be precisely determined. However, reasonable estimates of lead-poisoning losses in different species can be made on the basis of waterfowl mortality reports and gizzard analyses. Within the United States, annual losses from lead poisoning have been estimated at between 1.6 and 2.4 million waterfowl, based on a fall flight of 100 million birds. Proportional adjustments that reflect current waterfowl populations and increasing use of nontoxic shot should be made when estimating current lead poisoning losses.

Lead poisoning is common in mallard, northern pintail, redhead, scaup, Canada and snow geese, and tundra swan. The frequency of this disease decreases with increasing specialization of food habits and higher percentages of fish in the diet. Therefore, goldeneye are seldom affected and mergansers rarely affected (Fig. 18.1). Among land birds, eagles are most frequently reported dying from lead poisoning (Fig. 18.2). Lead poisoning in eagles generally is a result of swallowing lead shot embedded in the flesh of their prey.

Distribution

Losses occur coast-to-coast and border-to-border within the United States. Documented occurrences of lead poisoning in migratory birds vary widely between States and do not necessarily reflect true geographic differences in the frequency of occurrence of this condition. For example, although the geographic distribution of lead poisoning in bald eagles is closely associated with their wintering areas, the number of lead poisoning cases from Wisconsin and Minnesota is disproportionately high. These data reflect minimum numbers of lead-poisoned bald eagles in the United States. Because submission of bald eagles for examination is variable, no direct comparison can be made between States regarding the number of lead-poisoned eagles (Fig. 18.3*a*). The reported distribution of lead poisoning in eagles as well as in waterfowl is more a function of recognition than of frequency of occurrence. The general distribution of this disease in waterfowl on the basis of lead shot ingestion surveys and documented mortality is reflected in Fig. 18.3*b*.

Lead poisoning has also been reported as a cause of migratory bird mortality in other countries, including Canada, Great Britain, Denmark, Sweden, Germany, Italy, and New Zealand.

Seasonality

Losses can occur at any time of the year, although most cases of lead poisoning occur after the waterfowl hunting season has been completed in northern areas and during the later part of the season in southern areas of the United States. January and February are peak months for cases in tundra swans, Canada geese, and puddle ducks. Spring losses are more commonly reported for diving ducks. Tundra swans are also frequently lead poisoned during spring migration.

Fig. 18.1. Relative occurrence of reported lead poisoning in North American waterfowl.

Type of bird	Relative occurrence
Ducks	
Puddle ducks	● ● ●
Bay diving ducks	● ● ●
Teal, shoveler, wood duck	●
Whistling ducks	○
Sea ducks	○
Mergansers	○
Geese	
Canada geese	● ● ●
Snow and Ross' geese	● ● ●
Brant and other geese	○
Swans	
Tundra swan	● ● ●
Mute swan	● ●
Other swans	●

● ● ● Frequent reports of mortality including individual die-offs involving hundreds to thousands of birds

● ● Often reported mortality

● Occasionally reported mortality; lead shot ingestion studies generally indicate low levels of exposure to lead shot

○ Rare reports of mortality; lead shot ingestion studies generally indicate little or no lead shot ingestion

Fig. 18.2. Relative occurrence of reported lead poisoning in nonwaterfowl species.

Type of bird	Relative occurrence
Water birds	
Shorebirds	●
Coots and rails	● ●
Cranes	●
Gulls	●
Other species	○
Upland game birds	
Pheasants and quail	●
Turkey and grouse	○
Doves	●
Woodcock	○
Raptors	
Bald eagles	● ● ●
Golden eagles	● ●
Hawks	●
Owls	○

● ● ● Frequent reports of mortality including individual die-offs involving hundreds to thousands of birds

● ● Often reported mortality

● Occasionally reported mortality; lead shot ingestion studies generally indicate low levels of exposure to lead shot

○ Rare reports of mortality; lead shot ingestion studies generally indicate little or no lead shot ingestion

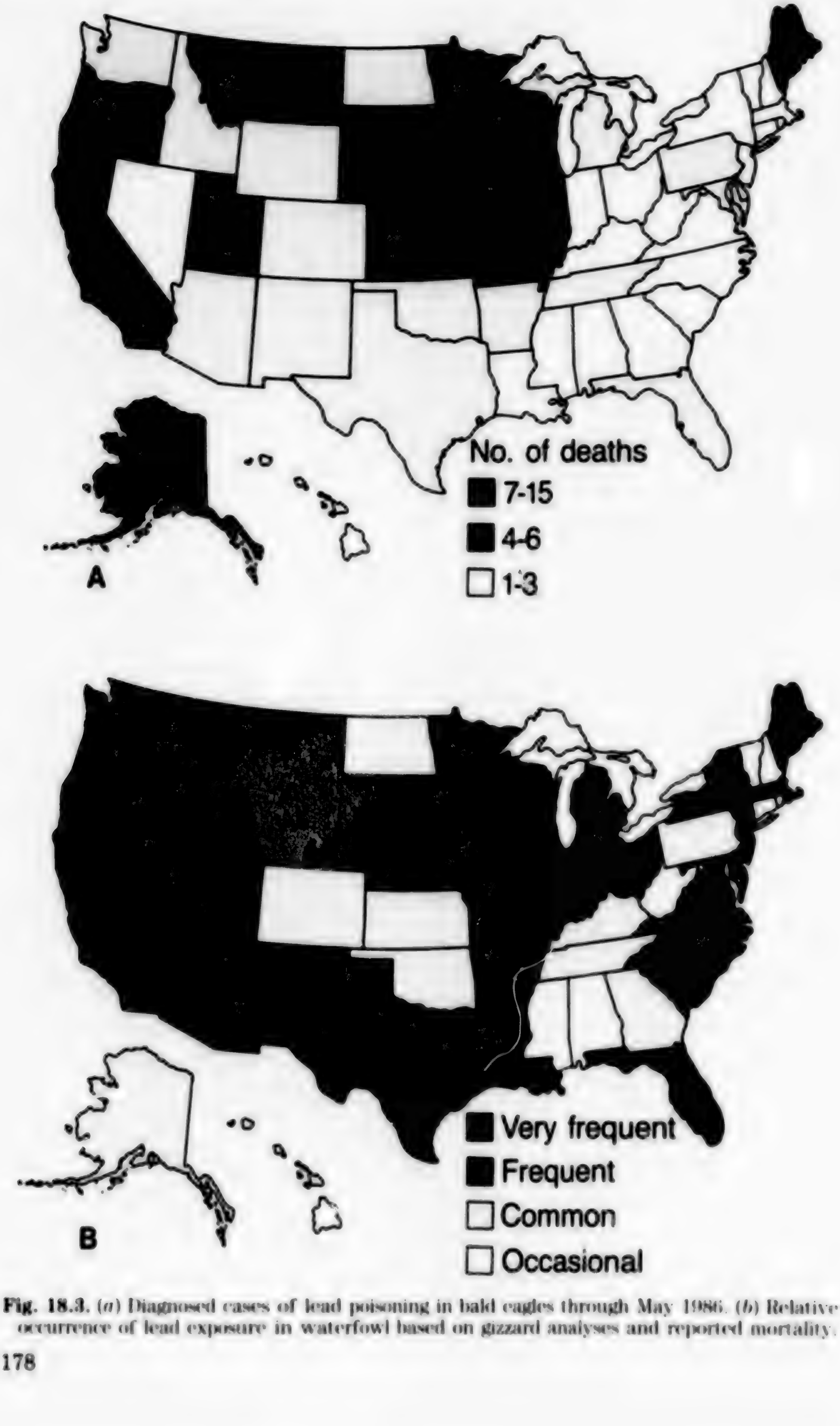


Fig. 18.3. (*a*) Diagnosed cases of lead poisoning in bald eagles through May 1986. (*b*) Relative occurrence of lead exposure in waterfowl based on gizzard analyses and reported mortality.

Field Signs

Lead-poisoned waterfowl are often mistaken for hunting season cripples. Special attention should be given to waterfowl that do not take flight when the flock is disturbed and to small aggregations of waterfowl that remain after most other birds of that species have migrated from the area. Lead-poisoned birds become reluctant to fly when approached; those that can still fly are often noticeably weak flyers, unable to sustain flight for any distance, flying erratically and landing poorly. Birds that attempt to escape pursuit by running may exhibit an unsteady gait. In lead-poisoned Canada geese, the head and neck position may appear "crooked" or bent in flight; a marked change in the tone of call is also sometimes evident in this species. As the disease progresses and waterfowl become flightless, the wings are held in a characteristic "roof-shaped" position (Fig. 18.4) followed by wing droop as the birds become increasingly moribund (Fig. 18.5). There may be a fluid discharge from the bill, and often there is an absence of escape response in the presence of man.

Lead-poisoned waterfowl are easily captured during advanced stages of intoxication (Fig. 18.6). Because severely affected birds generally seek isolation and protective cover, well-trained retrieving dogs can help greatly to locate and collect these birds. An abundance of bile-stained feces on an area used by waterfowl (Figs. 18.7*a* and *b*) is suggestive of lead poisoning and warrants ground searches even if other field signs have not been observed. Green-colored feces can also result from feeding on green wheat and other plants, but the coloration is somewhat different.

Gross Lesions

Lead-poisoned waterfowl are often emaciated because of the prolonged course of the illness and its impact on essential body processes. Therefore, many affected birds appear to be starving; they are light in weight, have a "hatchet-breast" appearance (Fig. 18.8), and the undersurface of their skin is devoid of fat (Fig. 18.9). The vent area of these birds is often stained with a bright green diarrhea (Fig. 18.10). The heads of Canada geese may appear puffy or swollen because serumlike fluids accumulate in the tissues of the face (Fig. 18.11).

Lesions observed at necropsy of lead-poisoned birds that have died after a prolonged illness generally consist of the following:

1. Severe wasting of the breast muscles (Fig. 18.8).
2. Absent or reduced amounts of visceral fat (Fig. 18.9).
3. Impactions of the esophagus or proventriculus in about 20–30% of affected waterfowl. These impactions may contain food items, or combinations of food, sand, and mud. The extent of impaction may be restricted to the gizzard and proventriculus, extend to the mouth, or lie somewhere in between (Fig. 18.12).
4. A prominent gallbladder that is distended, filled with bile, and dark or bright green (Fig. 18.13).
5. Normally yellow gizzard lining discolored a dark or bright green (Fig. 18.14). Gizzard contents are also often bile-stained.
6. Lead pellets or small particles of lead often present among gizzard and proventricular contents. Pellets that have been present for a long time are well worn, reduced in size, and disklike rather than spherical (Fig. 18.15). Careful washing of contents is required to find smaller lead fragments. X-ray examination is often used to detect radiopaque objects in gizzards, but recovery of the objects is necessary to separate lead from other metals. Flushing contents through a series of progressively smaller sieves is one method for pellet recovery.

Less obvious pathological changes include wasting of internal organs such as liver, kidneys, and spleen; areas of paleness in the heart muscle; flabby-looking heart; and paler-than-normal-looking internal organs and muscle tissue.

The above field signs and gross lesions provide a basis for a presumptive diagnosis of lead poisoning. However, none of these signs and lesions is diagnostic by itself and all can result from other causes. Also, many of the above signs and lesions are absent in birds that die acutely following an overwhelming lead exposure.

18.4*a*

18.4*b*

Fig. 18.4. Characteristic "roof-shaped" position of the wings in (*a*) a lead-poisoned mallard (leading bird) and (*b*) a snow goose. (*a*) *J.R.*; (*b*) *M.F.*

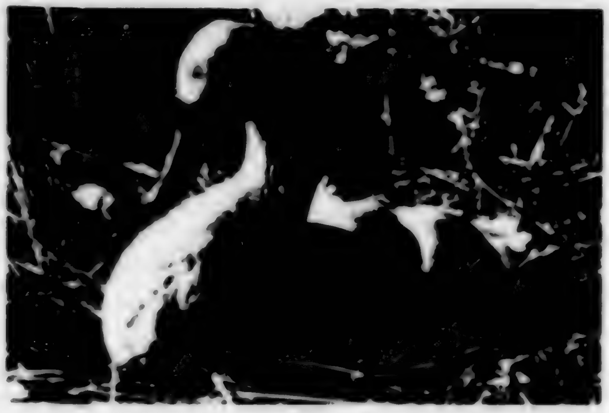
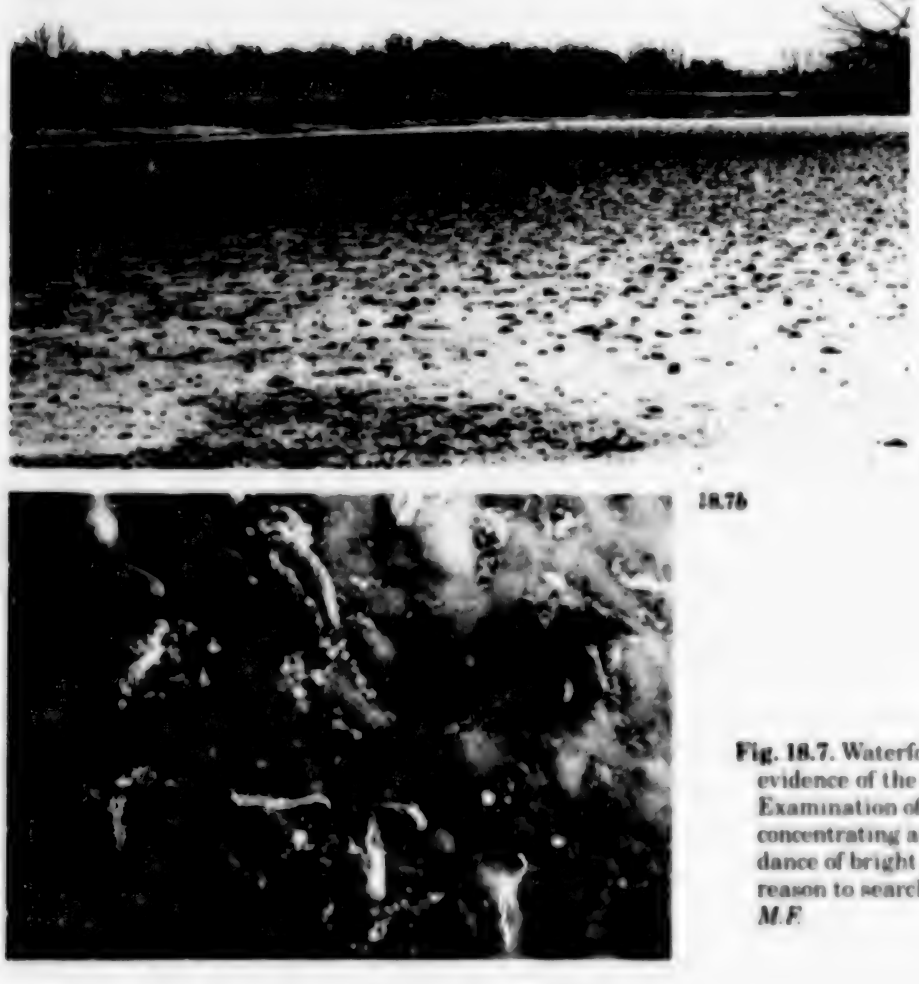


Fig. 18.5. Wing droop in a tundra swan in advanced stages of lead intoxication. *M.F.*

Fig. 18.6. Inability of these lead-poisoned Canada geese to escape capture by man illustrates their great vulnerability to predation. *M.F.*



18.7*a*

18.7*b*

Fig. 18.7. Waterfowl feces provide presumptive evidence of the occurrence of lead poisoning. Examination of (*a*) feces where waterfowl are concentrating and (*b*) observations of an abundance of bright green-colored feces should be reason to search for sick birds and carcasses. *M.F.*

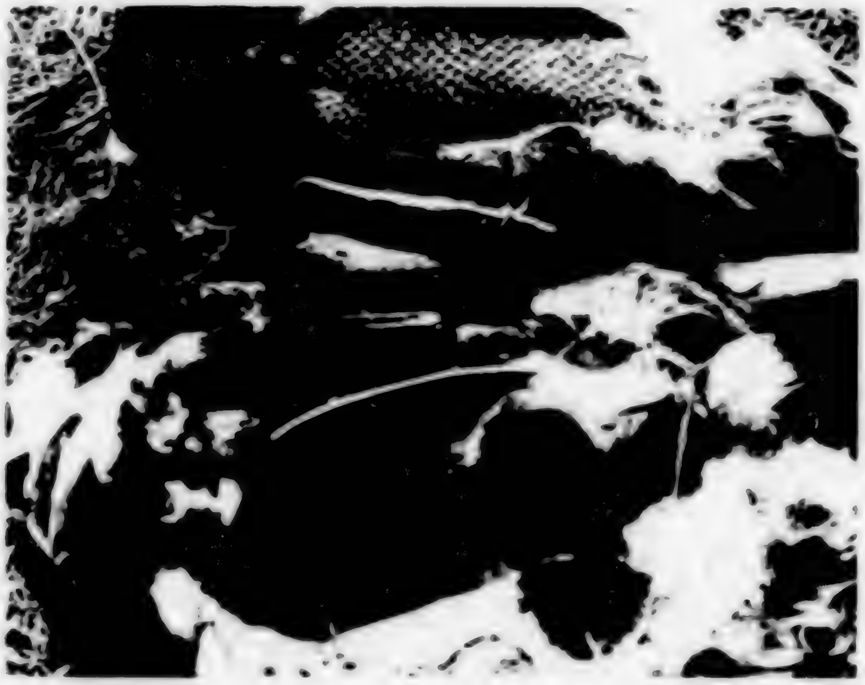
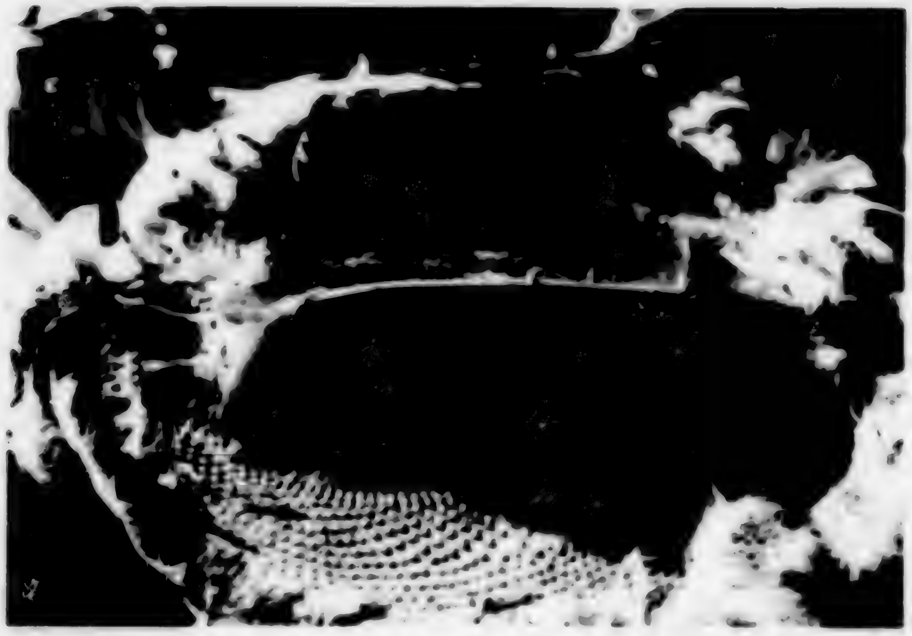


Fig. 18.8. "Hatchet-breast" appearance of a lead-poisoned mallard (top bird) and northern pintail. The skin has been removed from the breast of the pintail to further illustrate the severe loss of muscle tissue. *M.F.*

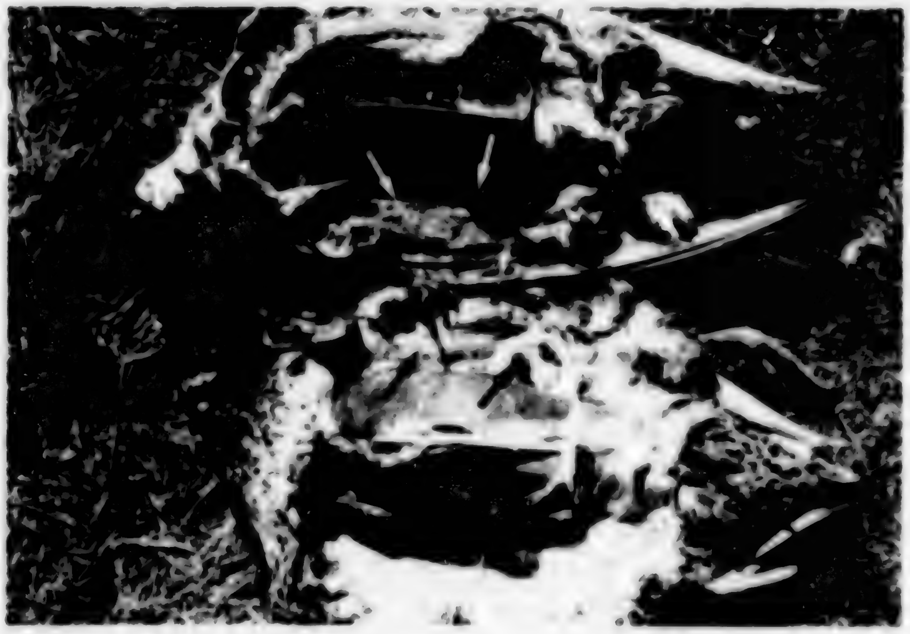
Diagnosis

A definitive diagnosis of lead poisoning is based on pathological and toxicological findings supplemented by clinical signs and field observations. The presence or absence of lead shot or lead particles in the gizzard contents is useful information and should be recorded, but it is not diagnostic. Liver or kidney are the tissues of choice for toxicology analysis, with liver tissue being more commonly used. If you suspect lead poisoning and cannot submit whole birds to the diagnostic laboratory, remove liver or kidney tissue, wrap separately in aluminum foil, and freeze until submitted for analysis. Collect the entire liver or one entire kidney. However, since toxicology is but one aspect of reaching a lead-poisoning diagnosis, make every effort to submit whole birds for analysis. Liver lead values of 6–8 ppm or higher on a wet-weight basis, or 20–30 ppm on a dry-weight basis, are suggestive of lead poisoning when other signs of lead poisoning are present.

Lead levels in populations of live birds can be evaluated by using whole blood. Collect a minimum of 2 to 5 mL of blood in lead-free tubes containing an anticoagulant such as sodium citrate or EDTA. Evidence of lead exposure can also be obtained through indirect measurements involving blood enzymes. Measurement of protoporphyrin IX in red blood cells is the most popular assay because only a few drops of blood are needed and testing is inexpensive once appropriate instrumentation is obtained. Elevated blood protoporphyrin levels are correlated with lead exposure and serve as a sensitive screening assay, but do not provide direct measurement of the amount of lead in blood. This technique has its greatest value in identifying populations from which more direct measurements should be taken, and for screening blood samples to determine which should be tested for blood lead concentrations. Confirm correct procedures for collecting blood samples for lead analysis with the diagnostic laboratory before samples are collected. Keep blood samples chilled until submitted for analysis, regardless of the assay being used. Write the date and time of collection on the tube.



18.9a



18.9b

Fig. 18.9. Loss of subcutaneous fat is often extreme in lead-poisoned birds. (*a*) The undersurface of the skin of this pintail is totally devoid of fat, in contrast with (*b*) the abundance of yellow fat present in the mallard (*bottom bird*) that had died of avian cholera. Note also the absence of fat in the visceral area and along the knees of the northern pintail (*top bird*) in comparison with the mallard. *M.F.*

Fig. 18.10. Bright green staining of the vent area is often indicative of lead poisoning. *M.F.*

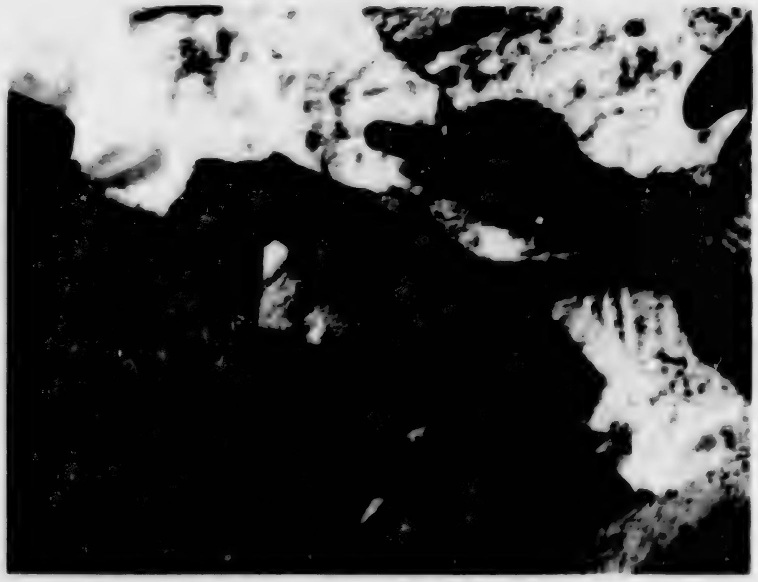


Fig. 18.11. The heads of lead-poisoned Canada geese often appear puffy or swollen. *M.F.*

18.12a

18.12b

Fig. 18.12. Examples of impactions in lead-poisoned birds. (*a*) Impaction of corn in digestive tract of a hen mallard, extending from the gizzard to the mouth; (*b*) goose with an impaction of grasses; *(continued on next page)*

18.12c

18.12*d*

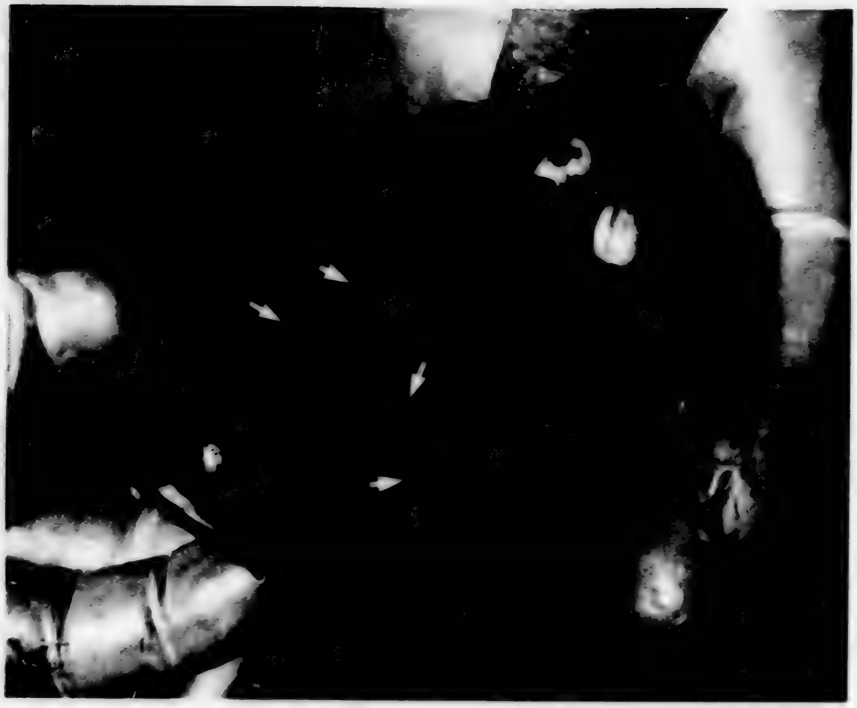
(*c*) tundra swan with an impaction of grasses and some seeds, extending from the mouth to the gizzard; and (*d*) a more limited impaction in a drake mallard. *M.F.*

Fig. 18.13. (*a*) The gallbladder of lead-poisoned birds is often distended and filled with bright green bile. (*b*) Note also the lead shot present in the gizzard of this bird. *M.F.*

18.14*a*

18.14*b*

Fig. 18.14. (a) Comparison between the appearance of the gizzard lining of a lead-poisoned mallard (*left*) and normal mallard (*right*). (*b*) Pathological changes in the gizzard of a lead-poisoned bird. Note green-stained coloration and hard appearance of tissue. The gizzard lining has split (*arrow*) because the tissue has become so brittle. Note also the presence of lead shot among the grit in the center of the pad. (*a*) *J.L.C.*; (*b*) *M.F.*



18.15a

18.15b

Fig. 18.15. (*a*) Presence of lead shot in Canada goose gizzard. Note presence of corn. Corn and other cereal grains intensify the toxicity of lead. (*b*) Lead shot, originally round, have been worn down in a waterfowl gizzard. Note the flattened, disk-like shape of many of these pellets. (*a*) *M.F.*; (*b*) *J.R.*

Control

Two actions can often be taken to reduce the magnitude of mortality from lead poisoning when die-offs occur; denying bird use in problem areas, and rigorous pickup and proper disposal of dead and moribund birds.

Denying birds use of problem areas requires knowing where the birds are picking up the lead. This is complicated by the fact that signs of intoxication may not appear until a week after lead ingestion, and birds may not start dying until 2 to 3 weeks after lead ingestion. Habitat modification is also useful in some instances, but differences in feeding habits must be considered. For example, placing additional water on an area may protect puddle ducks from reaching lead shot on the bottom of wetlands, but this creates attractive feeding areas for diving ducks. Similarly, draining an area may prevent ingestion of lead shot by waterfowl, but creates an attractive feeding area for shorebirds or pheasants. Therefore, control actions must consider the broad spectrum of wildlife likely to use the area at the time action is taken. Rigorous pickup and proper disposal of lead-contaminated waterfowl carcasses is required to prevent raptors and other scavenger species from ingesting them. The high percentage of waterfowl with embedded body shot provides a continual opportunity for lead exposure in raptors that far exceeds the opportunity for ingestion of shot present in waterfowl gizzards.

Other management practices that have been used to reduce losses from lead poisoning on site-specific areas include: (1) tillage programs to turn lead shot below the surface of soil so that shot is not readily available to birds; (2) planting food crops other than corn and other grains that aggravate the effects of lead ingestion; and (3) requiring the use of nontoxic shot on hunting areas. The potential contributions of the first two practices toward reducing lead-poisoning losses among migratory birds are, at best, limited and temporary. The use of nontoxic shot is the only long-term solution for significantly reducing migratory bird losses from lead poisoning.

Medical treatment of lead-poisoned birds is generally not a reasonable approach. However, endangered species or other birds of high individual value that are lead poisoned may warrant medical treatment. In those instances, treatment should be done only by qualified persons familiar with and skilled in the proper use of lead-chelating chemicals. Under the best of circumstances, results of treatment are unpredictable and the success rate low.

Human Health Considerations

People do inadvertently consume lead-poisoned waterfowl. Although this is not desirable, no appreciable risks to human health exist. Most lead present in the body of a lead-poisoned bird is in soft tissues such as liver and kidneys rather than in the flesh. The dose relation (mg of lead per kg of body weight) and lead excretion processes are such that a great number of lead-poisoned birds would need to be consumed in a relatively short time before toxic levels of lead could build up in the human body. Persons who eat liver, kidney, and other soft tissues from lead-poisoned birds would consume more lead than those who eat only muscle tissue of these birds. Persons who consume waterfowl bone marrow would be additionally exposed to lead, since lead is stored long-term in bone.

There are a few documented cases of humans developing lead poisoning after having accidentally ingested lead shot embedded in the meat they ate. This type of lead poisoning is rare, perhaps due to caution exercised when eating hunter-killed wildlife so as to avoid potential damage to teeth from biting into shot. Lead shot that is ingested can also become lodged in the appendix, resulting in appendicitis. Although this does not happen often, it happens most in people who hunt waterfowl for subsistence.

Supplementary Reading

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Chapter 19.
Oil Toxicosis

Patricia A. Gullett

Synonyms

None

Cause

Birds exposed to spilled petroleum products suffer from hypothermia as a result of physical contamination, and also from poisoning due to ingestion, inhalation, or absorption of the product.

Natural oil seeps in the Santa Barbara Channel, California, claim hundreds, if not thousands, of sea birds annually, but human-caused oil spills are more widely distributed and more damaging to bird populations.

An average of 15 million gallons of petroleum products are spilled yearly in and around the United States. Most (42%) are spilled during transport over water; another 20% are spilled during land and pipeline transport (Fig. 19.1).

Migratory bird exposure to oil also occurs from discharges of oil-laden wastewater into settling ponds and wetlands.

Species Affected

A wide variety of migratory birds and other wildlife have been affected by oil (Fig. 19.2). The type of animal affected depends on the location of the spill and the bird's behavior.

Many sea birds such as murres and puffins have a low reproductive rate and may require decades to rebuild their populations to prespill numbers. Species with high reproductive rates recover from a spill quickly. Endangered species such as brown pelicans are vulnerable to catastrophic losses.

Distribution

Migratory bird exposure to oil is not limited to specific geographic areas. Oil spills have occurred in all 50 States. About one-third (Fig. 19.3) occur in inland areas. More than half (55%) of the spills occur in river channels as opposed to coastal and ocean areas (35%).

Other opportunities for exposure to oil occur in association with oil production, petroleum refining, and highly industrialized locations within the United States.

Seasonality

Winter storms increase the likelihood of transport spills, making January, February, and March the peak spill season. This is also the time of year when sea birds and waterfowl congregate in wintering areas, resulting in the potential for significant bird losses.

Field Signs

Birds frequently look wet and chilled because oiling damages feather waterproofing and insulating properties. Oiled birds may appear to be wet and chilled and may ride lower in the water than normal because they have lost feather buoyancy. Oiling is suggested when unusual numbers of water birds leave the water for islands, rocks, pilings, and other surfaces because of chilling. Birds that survive for 48 hours or more after oiling are often thin

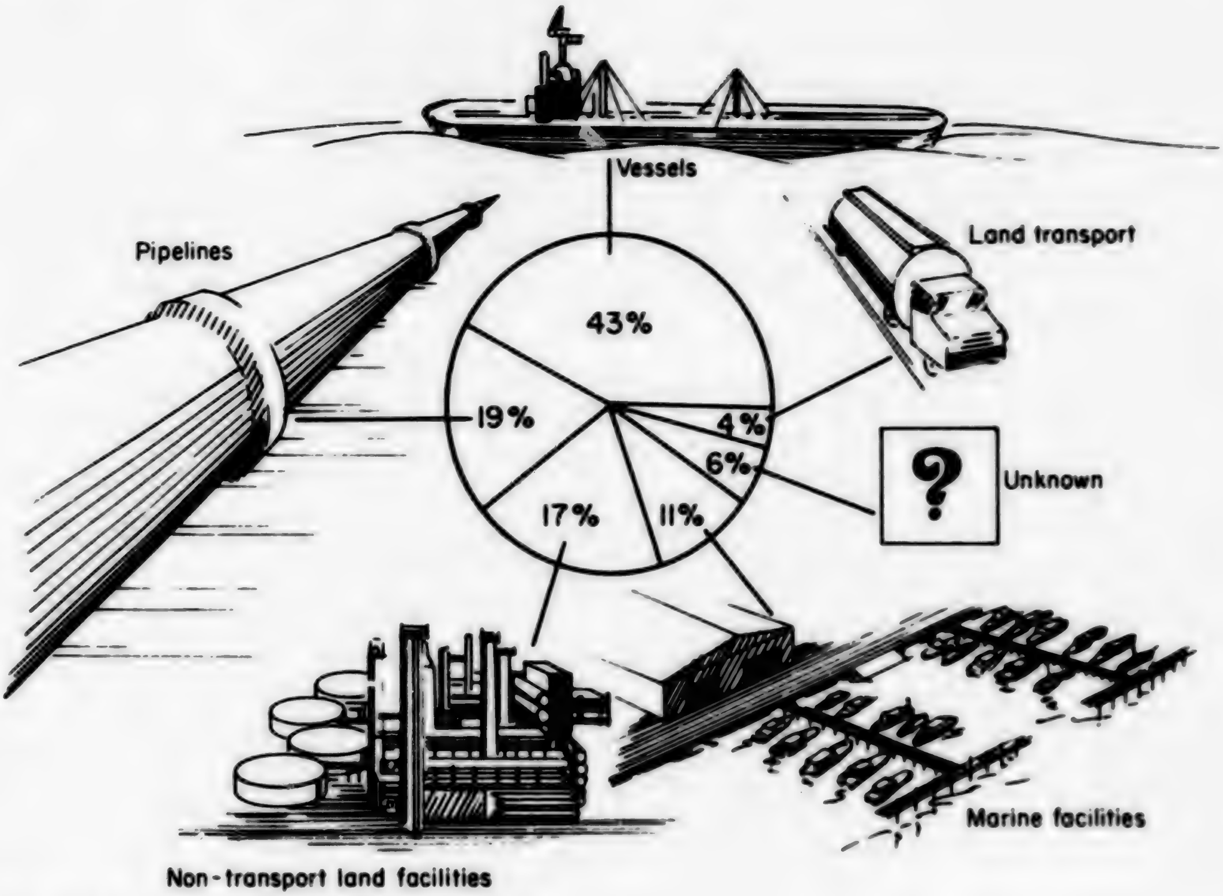


Fig. 19.1. Frequency of various kinds of oil spills.

and even close to starvation because they have stopped feeding and are utilizing body fat and then muscle tissue to produce heat in response to chilling.

Oil can usually be seen or smelled on the feathers, but some light, transparent oils may be difficult to detect. One useful technique for detecting oiling is to place a few feathers from the bird in a pan of water and watch for an oil sheen to appear.

Gross Lesions

Necropsy findings of birds that die from oil exposure are highly variable. Birds are often emaciated and oil might be present in the trachea, lungs, digestive tract, and around the vent. The lining of the intestine may be reddened or the intestine can contain blood. The salt glands (located over the eyes) might appear swollen and the adrenal glands can be enlarged. A variety of other changes in the normal appearance of tissues and organs may be present.

Diagnosis

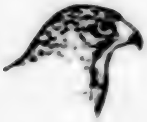
Diagnosis of oiling is seldom a problem. Seeing oil on the bird or in the environment usually suffices (Fig. 19.4). Major oil spills are frequently well publicized by intensive media coverage before slicks or affected birds appear. However, small spills, especially those of unknown origin, often go unnoticed except for the appearance of a few contaminated birds. After the spill is cleaned or

Fig. 19.2. Susceptibility to oiling in major groups of wild birds.

Type of bird	Relative susceptibility
Ducks, geese, and swans	● ● ●
Sea and bay ducks	● ● ●
Grebes	● ● ●
Loons	● ● ●
Cormorants	● ●
Cranes	●
Waders (herons, egrets, bitterns)	● ●
Plovers, sandpipers	●
Gulls	● ●
Pelicans	● ● ●

● ● ● High susceptibility
● ● Medium susceptibility
● Low susceptibility

Fig. 19.2. Susceptibility to oiling in major groups of wild birds. (Continued)

Type of bird		Relative susceptibility
Pelagic birds (albatross, petrel, shearwater, fulmar, frigate)		●
Alcids (murres, puffins)		● ● ●
Raptors		●
Songbirds		●

● ● ● High susceptibility
● ● Medium susceptibility
● Low susceptibility

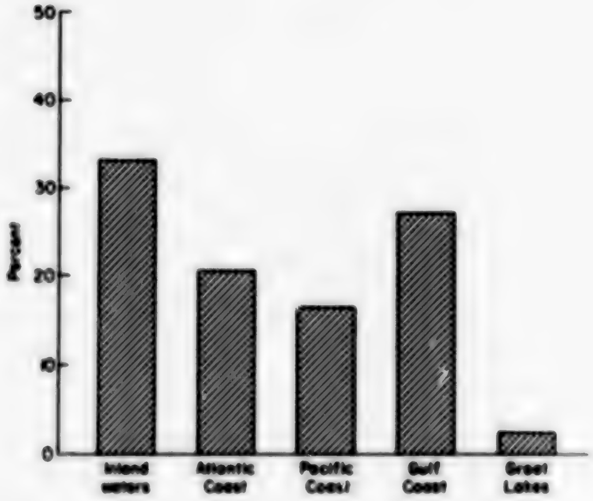


Fig. 19.3. Distribution of spilled oil, 1978–83.

Fig. 19.4. Oiled great blue heron. *J.R.*

dispersed, chronic losses can result by birds ingesting oil in food items. Subtle effects on reproduction, such as decreased egg production, reduced fertility and hatchability, and decreased sperm production can all occur. Chemical analysis of tissues or eggs cannot be used for diagnostic purposes because petroleum products have a complex chemical composition. Therefore, good background information and field observations are an integral part of specimen submission to diagnostic laboratories (see chapter on Recording and Submitting Specimen History Data).

Submit whole carcasses whenever possible. If this cannot be done and oiling is suspected as the cause of death, remove the lungs intact and place them in a 10% buffered formalin solution. Microscopic examination of this tissue can be useful in diagnosing death caused by inhalation of oil. Do not freeze formalin-fixed tissues.

Control

In the event of a spill, contact the National Response Center at the 24-hour, toll-free number (800)424-8802. They will advise the responsible agency (Coast Guard or Environmental Protection Agency) which will then respond to the event.

Scaring devices and other forms of disturbance can be used to discourage bird use of the problem area. If this area is being used or is likely to be used by endangered species, it may be helpful to initiate actions that will attract the birds to other locations while the spill is contained and cleaned.

Cleaning oiled birds is perhaps not justified on a "population" basis, but is desired by the public and warranted when rare, threatened, or endangered species are involved. These activities *should not be attempted* without knowledge of cleaning techniques. Advice can be obtained from most wildlife resource agencies. The cost of cleaning birds is sometimes, but not always, assumed by the party responsible for the spill.

Human Health Considerations

Direct contact with the oil and activities associated with the cleanup are potentially hazardous to humans. Petroleum products may be contaminated by a host of toxins that include PCB's (polychlorinated biphenyls) and organophosphates. Wear protective clothing to prevent direct exposure of skin surfaces to oil. Preventing injuries during spill containment and cleanup requires levelheadedness, advice from experts, and close supervision of workers — especially volunteers. Two major concerns are drowning and hypothermia.

Workers should not enter the water or climb slippery cliffs to rescue birds since the birds themselves present a hazard. Many sea birds have sharp, "spearing" beaks and often aim for the eyes of their predators—and their caretakers! Workers should wear goggles.

Supplementary Reading

International Bird Rescue Research Center. 1978. Saving oiled seabirds; a manual for cleaning and rehabilitating oiled waterfowl. American Petroleum Institute, 2101 L St. N.W., Washington, D.C. 20037. 35 pp.

Fletcher, A. 1977. Status and future trends in oil spills and implications for the FWS *in* P. L. Fore, editor. Proceedings of the 1977 oil spills response workshop. Cosponsored by Office of Migratory Bird Management and Office of Biological Services, U.S. Fish and Wildlife Service, Washington, D.C. 153 pp.

U.S. Fish and Wildlife Service. 1977. Oiled bird collection and care. Appendix 4. *in* USFWS pollution response plan for oil and hazardous substances. Office of Migratory Bird Management and Office of the Coordinator, Environmental Contaminants Evaluation Program, U.S. Fish and Wildlife Service, Washington, D.C.

Appendix A.

Sample Specimen History Form

Submitter's name:

Affiliation:

Address:

Telephone:

Date collected:

Collector's name:

Specific die-off location:

State:

County:

Environmental factors: (Record such conditions as storms, precipitation, temperature changes, or other changes that may contribute to stress.)

Disease onset: (Your best estimate of when the outbreak started.)

Species affected: (The diversity of species affected may provide clues to the disease involved.)

Age/sex: (Any selective mortality related to age and sex.)

Morbidity/mortality: (Ratio of sick animals to dead animals.)

Known dead: (Actual pickup figures.)

Estimated dead: (Consider removal by scavengers or other means.)

Clinical signs: (Any unusual behavior and physical appearance.)

Population at risk: (Number of animals in the area that could be exposed to the disease.)

Population movement: (Recent changes in the number of animals on the area and their source or destination, if known.)

Problem area description: (Land use, habitat types, and other distinctive features.)

Comments:

Appendix B.

Sources of Wildlife Disease Diagnostic Assistance in the United States

Assistance in obtaining a diagnosis of wildlife illness or death is available from a variety of sources. However, the ability of these sources to help you in a timely and effective way is influenced by the primary mission of that source and its administrative flexibility and constraints. Therefore, it is advisable to make inquiries before the need arises about available services; general response time for completing work submitted; processing costs, if any; who pays shipping costs; and who to contact when assistance is required.

The following wildlife disease programs can offer information and various types of assistance and services.

Wildlife Disease Programs

1. *U.S. Fish and Wildlife Service, National Wildlife Health Center, Madison, Wisconsin* (fish disease work is handled by other Fish and Wildlife Service facilities).

2. *State fish and game agencies.* Several States have active wildlife disease programs, located at: Fairbanks, Alaska; Sacramento, California; Fort Collins, Colorado; Rose Lake, Michigan; Hampton, New Jersey; Delmar, New York; Fargo, North Dakota; Madison Wisconsin; and Laramie, Wyoming.

3. *Regional wildlife disease programs.* Three regional programs are presently affiliated with universities:
   - a. Southeastern Cooperative Wildlife Disease Study, University of Georgia-Athens.
   - b. Northeastern Center for Wildlife Disease, University of Connecticut-Storrs.
   - c. Colorado Wild Animal Disease Center, Colorado State University-Fort Collins.

4. *University programs.* Several universities, in addition to those housing regional wildlife disease programs, are actively involved in wildlife disease activities.

The University of Florida–Gainesville and Virginia Polytechnical Institute and State University–Blacksburg are two of the more active. Inquiries at Schools of Veterinary Medicine and Departments of Veterinary or Animal Science throughout the United States will reveal additional sources of wildlife disease diagnostic assistance.

5. *Private sector.* Private consultants also deal with wildlife disease problems. Assistance provided by these sources is usually on a fee basis.

Animal Disease Programs

1. *U.S. Department of Agriculture, Veterinary Services Laboratory, Ames, Iowa.* Except under unusual circumstances, this program is unable to accept diagnostic specimens that have not been referred through appropriate channels.

2. *State departments of agriculture.* Animal disease diagnostic laboratories exist to serve domestic animal needs, but will often accept wildlife specimens.

3. *Private sector.* A number of veterinarians in private practice have both interest and expertise in diseases of wildlife and become involved with these problems on a volunteer or fee basis.

The above listing is not intended to be comprehensive. Rather, it illustrates the diversity of possible sources of assistance. Individual circumstances and events dictate which of these sources will be most useful to you.

Appendix C.

Sources of Supplies Used in Collecting, Preserving, and Shipping Specimens

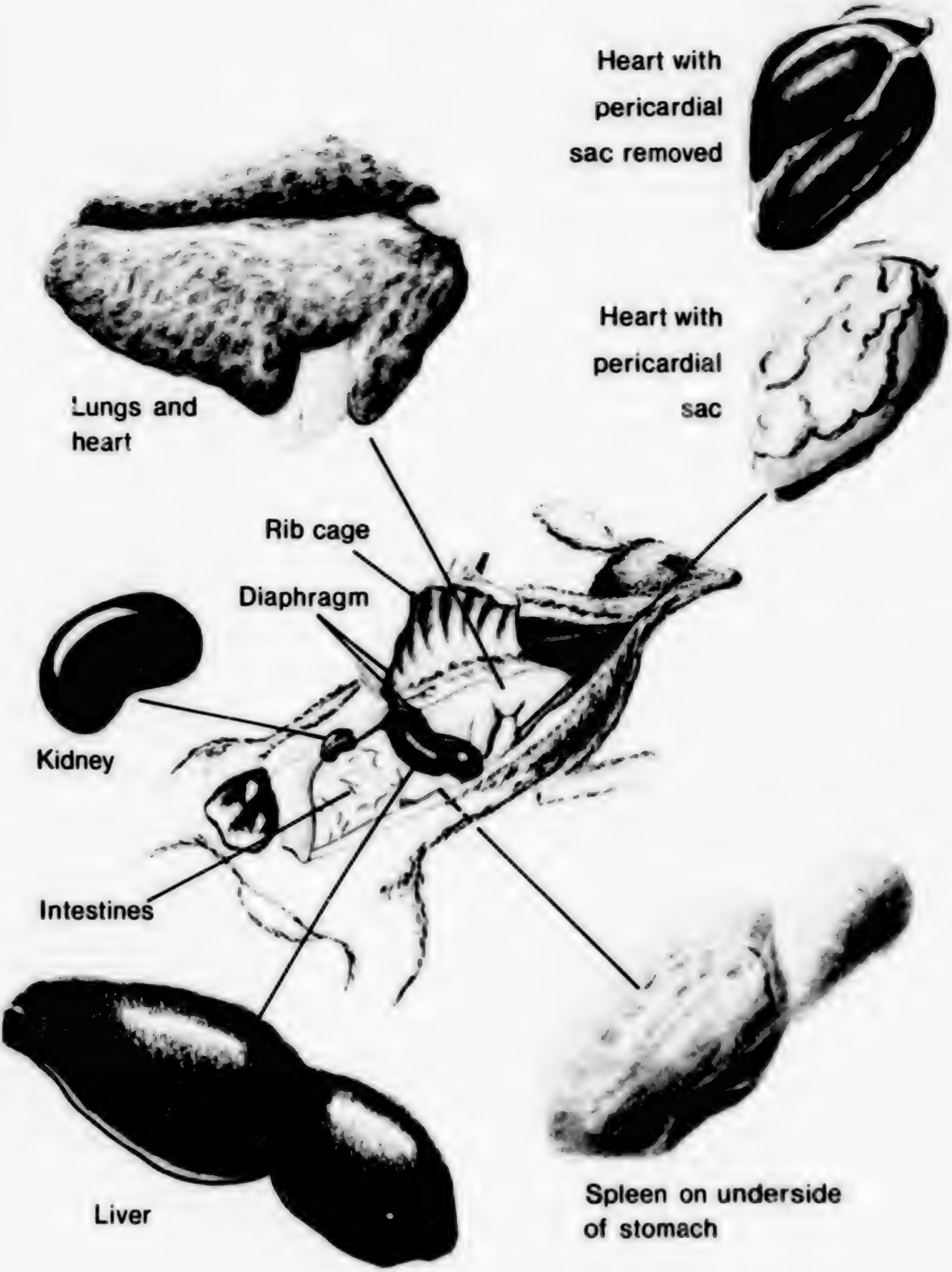
Company	Address and telephone	Item
American Scientific Products	Chicago Area Office 1210 Waukegan Road McGaw Park, IL 60085 Tel: (800)323-4515 or (800)942-4591	• Whirl-Pak bags • formalin • wide-mouth plastic jars
Curtis Matheson Scientific, Inc.	Chicago Area Branch 1850 Greenleaf Avenue Elk Grove Village, IL 60007 Tel: (312)439-5880	• Whirl-Pak bags • wide-mouth plastic jars
Fisher Scientific Co.	Chicago Regional Branch 1600 W. Glenlake Avenue Itaska, IL 60143 Tel: (312)773-3050	• Whirl-Pak bags • formalin
Thomas Scientific	Main Office 99 High Hill Road at I-295 P.O. Box 99 Swedesboro, NJ 08085-0090 Tel: (609)467-2000	• Whirl-Pak bags • wide-mouth plastic jars
VWR Scientific	P.O. Box 66929 O'Hare Amp Chicago, IL 60666 Tel: (800)932-5000	• Whirl-Pak bags
Local hospital and medical supply businesses		• Whirl-Pak bags • wide-mouth plastic jars
Some pharmacies		• formalin

Company	Address and telephone	Item
Freund Can Co.	151 W. 84th Street Chicago, IL 60620 Tel: (312)224-4300	• metal paint cans with lids
U.S. General Services Administration (GSA) (gov't employees and agencies only)	(1) General Services Admin. (Region 5) Customer Service Bureau (5FB) 230 S. Dearborn Street Chicago, IL 60604 Tel: FTS 353-5518 or FTS 886-9393 (quick order)	• strapping or "filament" tape
	(2) General Services Admin. Customer Service Bureau (8FB) Denver, CO 80225 Tel: (303)236-0018 or FTS 776-0018 (quick order)	
Local hardware, sports, and discount stores		• styrofoam shipping coolers • indelible markers • blue-ice paks
Sanford Corporation	Bellwood, IL 60104 Tel: (312)547-6650	• "Sharpie" markers
Polyfoam Packers Corp.	2320 South Foster Avenue Wheeling, IL 60090 Tel: (312)398-0110 (800)323-2440	• biomedical shippers and mailers

Appendix D.

Mammalian Dissection Illustration and Information

Fig. D.6. Gross appearance and location of organs of major significance in deer. Heart (encased in pericardial sac and with sac removed) and lungs (light pink color and spongy appearance) are found forward of the diaphragm and under the rib cage. The deep purple to purple-reddish brown colored liver is the prominent organ just behind the diaphragm and forward of the four-compartmented stomach, and intestines. The bluish-gray colored spleen is a much thinner organ than the liver and is located on the underside (left side) of the stomach area (lateral and dorsal to rumen) and is not readily visible until the stomach is rolled over. This organ is proportionally much larger and of different shape than in birds. The dark purple paired kidneys lie along the spinal column and are also quite different in appearance than those of birds. Considerable variation exists in the shape of kidneys among different animal species.


Heart with pericardial sac removed
Lungs and heart
Heart with pericardial sac
Rib cage
Diaphragm
Kidney
Intestines
Liver
Spleen on underside of stomach

Appendix E.

Common and Scientific Names of Wildlife[1]

Birds—Aves

- Loons—Order Gaviiformes
  - Common loon (*Gavia immer*)
- Grebes—Order Podicipediformes
- Tubenoses—Order Procellariiformes
  - Albatrosses—Diomedeidae
    - Laysan albatross (*Diomedea immutabilis*)
  - Shearwaters—Procellariidae
    - Northern Fulmar (*Fulmarus glacialis*)
  - Storm petrels—Hydrobatidae
- Penguins—Order Sphenisciformes
- Pelicans and allies—Order Pelecaniformes
  - White-tailed tropicbird (*Phaethon lepturus*)
  - Red-tailed tropicbird (*Phaethon rubricauda*)
  - Boobies (*Sula* spp.)
  - Northern gannet (*Sula bassanus*)
  - Pelicans (*Pelecanus* spp.)
  - Brown pelican (*Pelecanus occidentalis*)
  - Cormorants (*Phalacrocorax* spp.)
  - Frigatebirds (*Fregata* spp.)
- Herons and allies—Order Ciconiiformes
  - Bitterns—Botaurini
  - Herons—Ardeini
    - Great blue heron (*Ardea herodias*)
    - Egrets (*Casmerodius albus*, *Egretta* spp., *Bubulcus ibis*)
    - Great egret (*Casmerodius albus*)
    - Snowy egret (*Egretta thula*)
- Ibises and spoonbills—Threskiornithidae
  - Ibises (*Eudocimus* spp., *Plegadis* spp.)
  - Roseate spoonbill (*Ajaia ajaja*)
- Flamingos—Order Phoenicopteridae
  - Flamingos (*Phoenicopterus* spp.)
- Waterfowl—Order Anseriformes
  - Whistling ducks (*Dendrocygna* spp.)
  - Swans—Cygnini
    - Tundra swan (*Cygnus columbianus*)
    - Trumpeter swan (*Cygnus buccinator*)
    - Mute swan (*Cygnus olor*)
    - Black swan (*Cygnus atratus*)
  - Geese—Anserini
    - Greater white-fronted goose (*Anser albifrons*)
    - Snow goose (*Chen caerulescens*)
    - Ross' goose (*Chen rossii*)
    - Brant (*Branta bernicla*)
    - Canada goose (*Branta canadensis*)
    - Hawaiian goose (*Nesochen sandvicensis*)
  - Ducks—Anatinae
    - Muscovy duck (*Cairina moschat*)
    - Wood duck (*Aix sponsa*)
    - Teal (*Anas* spp., in part)
    - Green-winged teal (*Anas crecca*)
    - American black duck (*Anas rubripes*)
    - Mallard (*Anas platyrhynchos*)
    - Northern pintail (*Anas acuta*)
    - Blue-winged teal (*Anas discors*)
    - Northern shoveler (*Anas clypeata*)
    - Gadwall (*Anas strepera*)
    - American wigeon (*Anas americana*)
    - Canvasback (*Aythya valisineria*)
    - Redhead (*Aythya americana*)
    - Ring-necked duck (Aythya collaris)
    - Scaup (*Aythya* spp., in part)
    - Lesser scaup (*Aythya affinis*)
    - Eiders (*Somateria* spp., *Polysticta stelleri*)
    - Harlequin duck (*Histrionicus histrionicus*)
    - Oldsquaw (*Clangula hyemalis*)
    - Scoters (*Melanitta* spp.)

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[1] Authorities used:
American Ornithologists' Union. 1983.
Checklist of North American birds. 6th edition. Allen Press, Inc., Lawrence, Kans. 877 pp.

Jones, J. K., Jr., D. C. Carter, H. H. Genoways, R. S. Hoffman, and D. W. Rice. 1982. Revised checklist of North American mammals north of Mexico, 1982. Occas. Pap. Texas Tech Univ. 80. 22 pp.

    - Surf scoter (*Melanitta perspicillata*)
    - White-winged scoter (*Melanitta fusca*)
    - Goldeneyes (*Bucephala clangula, B. islandica*)
    - Common goldeneye (*Bucephala clangula*)
    - Bufflehead (*Bucephala albeola*)
    - Mergansers (*Lophodytes cucullatus, Mergus* spp.)
    - Ruddy duck (*Oxyura jamaicensis*)
- Vultures, Hawks, and Falcons—Order Falconiformes
  - American vultures—Cathartidae
  - Eagles—Accipitrinae, in part
  - Hawk—Accipitrinae, in part
  - Kite—Accipitrinae, in part
    - Bald eagle (*Haliaeetus leucocephalus*)
    - European sparrowhawk (*Accipiter nisus*)
    - Red-tailed hawk (*Buteo jamaicensis*)
    - Rough-legged hawk (*Buteo lagopus*)
    - Red-shouldered hawk (*Buteo lineatus*)
    - Golden eagle (*Aquila chrysaetos*)
  - True falcons—Falconini
- Gallinaceous birds—Order Galliformes
  - Partridges—Perdicini
  - Pheasants—Phasianini
    - Common peafowl (*Pavo cristatus*)
  - Grouse—Tetraoninae
    - Ptarmigans (*Lagopus* spp.)
    - Prairie chickens (*Tympanuchus* spp.)
    - Wild turkey (*Meleagris gallopavo*)
  - Quail—Odontophorinae
    - Northern bobwhite (*Colinus virginianus*)
    - California quail (*Callipepla californica*)
  - Guineafowl—Numidinae
- Cranes and allies—Order Gruiformes
  - Rails—Rallidae, in part
    - American coot (*Fulica americana*)
  - Cranes—Gruidae
    - Sandhill crane (*Grus canadensis*)
    - Common crane (*Grus grus*)
    - Manchurian crane [Red-crowned] (*Grus japonensis*)
    - Siberian white crane (*Grus leucogeranus*)
    - Hooded crane (*Grus monacha*)
    - White-naped crane (*Grus vipio*)
    - Sarus crane (*Grus antigone*)
    - Brolga crane (*Grus rubicunda*)
    - Stanley (Blue) crane (*Anthropoides paradisea*)
    - Demoiselle crane (*Anthropoides virgo*)
    - East African crowned crane (*Balearica regulorum*)
    - Crowned crane (*Balearica pavonina*)
- Shorebird—Order Charadriiformes, in part
  - Plover—Recurvirostridae
    - Black-necked stilt (*Himantopus mexicanus*)
    - American avocet (*Recurvirostra americana*)
  - Sandpipers—Scolopacinae
    - Yellowlegs (*Tringa* spp., in part)
    - Curlews (*Numenius* spp.)
    - Sanderling (*Calidris alba*)
    - American woodcock (*Scolopax minor*)
    - Phalaropes (*Phalaropus* spp.)
  - Jaegers—Stercorariinae
  - Gulls—Larinae
    - Herring gull (*Larus argentatus*)
  - Terns—Sterninae
  - Alcids—Alcidae
    - Murres (*Uria* spp.)
    - Common murre (*Uria aalge*)
    - Razorbill (*Alca torda*)
    - Guillemots (*Cepphus* spp.)
    - Murrelets and Auklets (*Brachyramphus* spp. *Synthiliboramphus* spp.)
    - Rhinoceros auklet (*Cerorhinaca monocerata*)
    - Puffins (*Fratercula* spp.)
- Pigeons and Doves—Order Columbiformes
    - Rock dove [feral pigeon] (*Columba livia*)
    - White-winged dove (*Zenaida asiatica*)
    - Mourning dove (*Zenaida macroura*)
- Parrots, Parakeets and Macaws—Order Psittacinae, in part
    - King parrots (*Alisterus* spp.)
- Owls—Order Strigiformes
- Perching birds—Order Passeriformes
    - Crows (*Corvus* spp., in part)
    - Ravens (*Corvus* spp., in part)
    - Eurasian jay (*Garrulus glandarius*)
    - European starling (*Sturnus vulgaris*, in part)
  - Cardinals, Grosbeaks, and allies—Cardinalinae, in part
    - Northern cardinal (*Cardinalis cardinalis*)
  - Blackbirds—Icterinae, in part
    - Common grackle (*Quiscalus quiscula*)
    - Brown-headed cowbird (*Molothrus ater*)
  - Finches—Fringillidae
    - Pine siskin (*Carduelis pinus*)
    - American goldfinch (*Carduelis tristis*)
    - Canaries (*Serinus* spp.)
    - Evening grosbeak (*Coccothraustes vespertinus*)

- Old World Sparrows—Passeridae
  - House sparrow (*Passer domesticus*)
- Estrildad Finches—Estrildidae
  - Java sparrow (*Padda oryzivora*)

Mammals—Mammalia

- Pouched Mammals—Order Marsupialia
  - New World Opossums—Didelphidae
    - Virginia opossum (*Didelphis virginiana*)
- Pikas, Hares, Rabbit—Order Lagomorpha
  - Rabbits—Leporidae
- Rodents—Order Rodentia
  - Squirrels—Sciuridae
    - Chipmunks (*Tamias* spp.)
  - Mice—Cricetidae, in part
  - Voles—Cricetidae, in part
    - Muskrat (*Ondatra zibethicus*)
  - Old World Rats and Mice—Muridae
    - Rat (*Rattus spp.*)
  - Hutias and Nutria—Myocastoridae
  - Nutria (*Myocastor coypus*)
- Carnivores—Order Carnivora
  - Foxes—Canidae, in part
  - Bears—Ursidae
  - Raccoons—Procyonidae
    - Raccoon (*Procyon lotor*)
  - Weasels—Mustelidae, in part
    - Weasels (*Mustela* spp.)
    - Mink (*Mustela vison*)
    - Skunks (*Mephitis* spp.)
  - Cats—Felidae
    - Mountain lion [puma] (*Felis concolor*)
    - Lynx (*Felix lynx*)
    - Bobcat (*Felis rufus*)
    - African lion (*Felis leo*)
- Even-toed ungulates—Order Artiodactyla
  - Deer—Cervidae
    - Elk (*Cervus elaphus*)
    - White-tailed deer (*Odocoileus virginianus*)
    - Moose (*Alces alces*)
    - Caribou, reindeer (*Rangifer tarandus*)
  - Cows and Sheep—Bovidae
    - Bison (*Bison bison*)
    - Mountain sheep [big horn] (*Ovis canadensis*)

Glossary of Technical Terms

abdominal wall—the layers of muscles lying between the skin and the abdominal cavity.

absorption—to take in a substance through the pores or cells of a tissue. The substance must pass through the tissue to be absorbed.

acute—sharp or severe; an acute illness is one having a short and relatively severe course.

ambient temperature—room or environmental temperature.

amyloid deposit—amyloid is a complex protein material that grossly resembles starch and in certain abnormal conditions accumulates in various body tissues causing cellular damage and injury to the affected organ.

anesthetic—a drug used to temporarily deaden pain.

anesthetic induction time—the time between administering an anesthetic chemical and the actual time when target nerves are deadened.

antigen—any foreign substance (generally proteins) to which the body reacts by producing antibodies. Antigens may be soluble substances such as toxins, particulate matter such as pollen, or microorganisms such as bacteria and viruses.

asymptomatic—without visible evidence of illness.

bacteriophage—a virus that attacks bacteria.

bacterium—singular for bacteria. Any of a group of microscopic, unicellular organisms with distinct cell membranes and lacking a distinct nucleus surrounded by a nuclear membrane.

barbiturate—a type of sedative or anesthetic, chemically derived from barbituric acid.

bile—yellow-brown to greenish liquid secreted by the liver and stored in the gallbladder before excretion by way of the intestine. It is composed of metabolic breakdown products derived from hemoglobin and other metabolic waste products.

broadcast—To spread widely, as when seeds are thrown out by hand.

buffered formalin—a 3.7% solution of formal (= 10% formalin) to which sodium phosphate buffers have been added. Best overall fixative for tissue for later microscopic study.

caecum (British; plural caeca) or cecum (American; plural ceca)—a large blind pouch or sac (often a pair) at the junction of the small intestine and large intestine.

calcification (of muscle tissue)—abnormal deposits of calcium salts in sites with dead tissues, parasites, or abnormal circulation.

central nervous system—the brain and spinal cord.

chelating chemical—a chemical that combines with a metal ion in a firm, ring-like band and prevents the metallic ion from having any further biochemical effect.

clinical sign—abnormal physiological change or behavior pattern that is indicative of illness. Signs are externally observable, as contrasted with symptoms, which are subjective. Therefore, only humans have symptoms.

cloaca— a common passage for fecal, urinary, and reproductive discharges in most lower vertebrates (birds, reptiles, and amphibians).

colibacillosis—infection with the bacterium *Escherichia coli*.

congenital abnormality—usually an anatomical malformation resulting from incomplete growth during embryonic development. Also, refers to an abnormal biochemical pathway caused by a genetic factor.

contagious—an infectious disease that is easily spread from one host to another host.

colon—the large intestine.

cornea—the transparent tissue on the front of the eyeball, specifically, covering the iris and pupil through which light passes to the interior.

coronary band—a fatty band encircling the heart; in hooved animals, the germinal layer beneath skin at the junction of the skin and hoof.

cosmopolitan—widely distributed throughout the world.

cracker shell—shotgun shell loaded to produce a loud sound in order to frighten animals.

cytoplasm—the aqueous portion of the cell outside the nucleus but contained within the cell wall. It is the site of most of the chemical activities of the cell.

definitive host—the host in which the adult parasite reproduces.

depopulation—the destruction of an exposed or infected group of animals.

desiccation—the act or process of drying a substance.

ecology—the study of the interrelations between living organisms and their environment.

emasculatome—instrument used to castrate male animals.

epizootiology—study of the effects of diseases on populations of animals.

erosion—wearing away; gradual disintegration.

etiological agent—any living or nonliving thing, power, or substance capable of causing a disease.

exsanguination (bleeding out)—the draining of blood from an animal.

"feather edge"—a long, shallow edge of a body of water that gradually deepens as one goes offshore.

femur—the thigh bone of humans; upper legbone in hooved mammals and birds. The bone between the pelvis and the knee.

fibrinous—composed of fibrinlike material; fibrin is an insoluble elastic protein in the serum that is involved in formation of clots.

gizzard—the enlarged muscular ventriculus ("second stomach") in many seed-eating birds.

"hatchet-breast"—a layman's term to describe the prominent, protruding breast keel seen as the result of the atrophy of the breast muscles. "The keel appears as sharp and as prominent as the blade of a hatchet."

heterogenous organism – one that is derived from a combination of different types of parent organisms.

herpesvirus—one of the major groups of related viruses with DNA nucleic acids and further characterized by similar size, shape, and physiochemical reactions.

husbandry practice—care and maintenance of animals.

hypersensitivity—greater than normal sensitivity to stimuli or to biological agents.

hypothermia—abnormal decreases in body temperature.

immunosuppressive therapy—medical treatment that suppresses the normal immune response.

impaction—an abnormal accumulation of food or other ingested materials that become lodged in a section of the digestive tract.

inapparent—an inapparent infection is one in which the infectious agent exists with the host but causes no recognizable signs of illness; the infectious agent may or may not be shed at irregular times.

infectious agent—a living organism capable of invading another; usually used when speaking of bacteria and viruses.

intermediate host—a host in which the parasite undergoes the nonreproductive phase of its life cycle.

intracellular parasite—parasitic organism, usually microscopic in size, that lives within the cells of the host animal.

joint capsule—the thick, fibrous capsule surrounding a joint, as around the knee.

lacrimal discharge—a discharge from the tear glands near the eye.

latent—a disease or infection not yet manifest by recognizable signs or lesions.

lesion – an abnormal change in tissue or organ due to disease or injury.

motility/motile/nonmotile—when discussing bacteria, terms refer to whether or not an organism moves on a particular culture medium; such motility reflects the presence of flagellae. Thus, absence or presence of motility is a useful characteristic in identifying bacteria.

morbidity—state of being sick. In actual use, the ratio of sick individuals to healthy individuals in a community or population.

moribund—"at the point of death." The animal is so sick that it will probably die soon.

mucous membrane—layer of tissue that lines a cavity or the intestinal tract and secretes a mixture of salts, sloughed cells, white blood cells, and proteins.

mucosal surface—a layer of cells lining the inside of the intestinal tract or other body part that secretes mucus.

mycotoxin—poison produced by various species of molds (fungi).

necropsy—postmortem examination of an animal.

necrosis—death of cells in an organ or tissue.

nictitating membrane—an inner eyelid (third eyelid) of birds, reptiles, and some mammals.

nodule—a small mass of tissue which is firm, discrete, and detectable by touch.

occlusion—the closure of teeth.

pathogenic—the ability to cause disease.

pathological—an adjective used to describe structural or functional changes that have occurred as the result of a disease.

pericardium—the fibrous sac surrounding the heart.

plaque—a patch or flat area; often fungi grow in flat spreading colonies called plaques.

postmortem—after death. A postmortem change is one that occurs after the death of the animal.

prevalence—the number of cases of a disease occurring at a particular time in a designated or defined area.

protozoan—one-celled animal with a recognizable nucleus, cytoplasm, and cytoplasmic structures.

protoporphyrin IX—an early metabolic product originating during the synthesis of porphyrins during the formation of hemoglobin.

proventriculus—the first, or "glandular", stomach of a bird.

psittacines—birds belonging to the group composed of macaws, parrots, parakeets, and other closely related species.

rendering—a process by which animal carcasses are converted into fats and fertilizer.

Section 7 consulations—the Endangered Species Act requires discussion and evaluation of any proposed Federal activity, program, or permit that might affect an endangered species.

sedated—chemically quieted.

serotype—a subpopulation of a species of microorganisms that has the same antigenic properties.

serum—clear, yellowish fluid component of blood after red cells are removed.

sloughing—shedding of dead cells or dead tissue from living structures or tissues.

subcutaneous—under the skin.

thymus gland—a lymph-gland-like organ located in the neck or upper thoracic cavity involved in cellular immunity.

toxic—poisonous.

toxicological—adjective pertaining to the findings of a toxicologist; toxicological data or toxicological study.

ubiquitous—found everywhere.

unthrifty appearance—an expression used in animal husbandry to describe an animal that is unkempt and dirty. Usually there is soiling of hair or feathers by excrement.

vascular system—blood circulation system.

vegetative form—in bacteria, an active, growing, mutiplying stage of development as opposed to a "spore," or resistant resting stage.

virus shedding—discharge of virus from body openings by way of exudate, excrement, or other body wastes or discharges.

virulent—highly infectious and very pathogenic.

viscera—internal organs, particularly of the thoracic and abdominal cavities.

Glossary of Animal Terms

alcids—typically pelagic colonial nesting seabirds that feed on fish by diving under the water surface to pursue their prey. Species include auklets, guillemots, murres, murrelets, and puffins.

bay ducks—bay diving ducks.

bay diving ducks—typically, ducks that feed in deep bodies of water, usually in the coastal bays and deep lakes, and dive for their food. These include canvasback, goldeneyes, redhead, and scaup.

birds of prey—this term is synonymous with raptors and includes eagles, hawks, falcons, kites, and owls.

carnivores—refers to flesh-eating mammals in the Order Carnivora—e.g. dogs, skunks, weasels, cats, raccoons.

dabbling ducks—also referred to as puddle ducks. Ducks that feed on surface or in shallow water and do not dive for their food—e.g., mallard, American black duck, gadwall, American wigeon, northern pintail, northern shoveler, and teal.

diving ducks—synonymous with bay-diving ducks.

gallinaceous birds—Synonymous with upland game birds.

gulls—refers to all *Larus* and *Rissa* (Family Laridae). True gulls and kittiwakes.

Hawaiian forest birds—native and introduced avifauna of the forested areas of the Hawaiian Islands. These may include such species as sparrows, finches, cardinals, honeycreepers, and thrushes

livestock—domestic animals raised for food and fiber such as hogs, sheep, cattle, and horses, but not including birds.

marine birds—birds of the open ocean, typically pelagic, and often colonial nesters. Examples include alcids, shearwaters, storm-petrels, albatrosses, jaegers, gannets, boobies, and frigatebirds.

mergansers—ducks with long, narrow serrated-edged bills, feeding primarily on fish.

migratory birds—all birds listed under provisions of the Migratory Bird Treaty Act and published in the *Federal Register.*

poultry—domestic species raised primarily for food and fiber; chickens, domestic ducks and geese, and common turkeys.

puddle ducks—synonymous with dabbling or surface-feeding ducks.

raptors—birds of prey that feed on the flesh of others. For purposes of this publication, typical species are hawks, falcons, owls, and eagles.

ruminants—hooved mammals possessing a rumen; includes members of the deer and cattle families—deer, elk, sheep, mountain sheep, and moose.

scavenger species—birds that often or habitually feed on dead, sick, or injured prey. Includes crows, vultures, bald eagles, and gulls.

sea ducks—ducks that frequent open ocean, even though some species are often found on coastal bays, larger inland lakes, and in other inland waters. Species include oldsquaw, eiders, scoters, and harlequin duck. Other forms of diving ducks such as goldeneyes, buffleheads, and bay-diving ducks are excluded for purposes of this publication.

shorebirds—birds that feed at the edge of shallow water, along mudflats, and in shallow wetlands where water depths do not exceed a few inches. Typically these birds feed on invertebrates and include such species as American avocet, black-necked stilt, curlews, plovers, phalaropes, sandpipers, yellowlegs, and sanderling.

songbirds—small perching and singing birds that typically belong to the Order Passeriforme, including sparrows, finches, and cardinals.

upland game birds—nonmigratory birds found in terrestrial habitats. These include prairie-chickens, wild turkey, quails, pheasants, grouse, partridges, and ptarmigans. Doves and American woodcock are considered separately for purposes of this publication.

wading birds—long-necked, long-legged birds that feed by wading in wetlands and catching prey with their bills. Examples are egrets, herons, ibises, Roseate spoonbills, flamingos, and bitterns.

waterfowl—ducks, geese, and swans. Does not include American coot.

Index

Adrenal glands, enlarged 192
Air, gasping for 102
Airboat 29, 40
Air sacs, flattened yellow plaques in 147–149
Albatross, oil toxicosis in 194
Alcids 213
  chlamydiosis in 109
  oil toxicosis in 194
Alkali poisoning, see Avian botulism
All-terrain vehicle 27, 31, 46, 53
American goldfinch, avian salmonellosis in 101
Amidostomum 159
Amphibious vehicle 31
Amyloid deposits 97, 209
Anesthetic gas 61, 209
Animal disease programs (U.S.) 199
Animal relocation 40–45
Antitoxin, for avian botulism 92–93
Area closure 41
Arthritis 101
Asper, see Aspergillosis
Aspergillosis 97, 145–150, 165
Aspergillus, see Aspergillosis
Avian botulism 83–93
Avian cholera 2, 46, 69–82, 92, 119, 183
Avian diphtheria, see Avian pox
Avian pasteurellosis, see Avian cholera
Avian pox 135–141
Avian salmonellosis 101-106
Avian tuberculosis 95–99

Bald eagle
  avian pox in 135
  lead poisoning in 175, 177–178
Bay diving ducks, see Diving ducks
Bay ducks, see Diving ducks
Beuthanasia-D Special 61
Big game, *Pasteurella multocida* in 70
Bill
  bloody discharge from 119, 121
  fluid discharge from 179
  scratching at 165
  wartlike nodules on 137
Bird, dissection of 10–11
Bird feeder, backyard 102, 104, 135, 137
Birds of prey, see Raptors
Bittern, oil toxicosis in 193
Blackbird
  aspergillosis in 145
  avian salmonellosis in 101
Black duck
  aspergillosis in 145
  duck plague in 117
  sarcocystis in 153, 157
Black swan, aspergillosis in 145
Blindness 153
Blood sample 9, 16–17
Blue ice pack 13–15, 18
Body bag, plastic 27
Boots 23, 46, 53
Botulism, see Avian botulism
Brant goose, leading poisoning in 176
Breast muscles, wasting of 2, 179
Breathing, difficulty in 102, 137, 147–148, 165
Brolga crane, inclusion body disease of cranes in 130, 132
Brooder pneumonia, see Aspergillosis
Brown-headed cowbird, avian salmonellosis in 101
Bufflehead, nasal leeches in 165
Burial of carcass 27, 53
Burning pit 27
Burning platform 27

Call, change in tone of 179
Canada goose
  aspergillosis in 145
  avian cholera in 72, 74, 82
  avian pox in 135
  duck plague in 117–118, 123, 125
  gizzard worms in 159, 161
  lead poisoning in 175–176, 179, 181, 184, 188
  sarcocystis in 153
Canary, avian salmonellosis in 102
Cannon net 54
Canvasback, nasal leeches in 165

Index

Capture device 41, 54
Carbon dioxide 61–63
Carbon monoxide 61–63
Carcass
  collection of 7, 23–27, 51, 53
  disposal of 27–39, 51, 53
  labeling of 10
  lines of, coinciding with water's edge 86, 88
  preservation of 7–12
  search for 23–27
  shipment of 14–20
  transport to disposal site 23–32, 53
Carnivores 207, 213
  Pasteurella multocida in 70
  sarcocystis in 154, 158
Castor bean poisoning 89
Cervical dislocation 59, 60
Chelating chemical 209
Chemical coolant 13
Chemical euthanasia 60–63
Chiggers 108
Chills 191–192
Chlamydia psittaci, see Chlamydiosis
Chlamydiosis 107–113
Chlorine bleach 7, 46
Cholera, see Avian cholera
Circular swimming 72, 75, 122
Clostridium botulinum, see Avian botulism
Clothing, protective 23–28
Cold sore lesion 119, 121
Colibacillosis 192
Collection of specimens 7–12, 200–201
Commercial carrier 20
Common crane, inclusion body disease of cranes in 130–132
Congenital abnormality 210
Conservation organization 46
Contagious disease 210
Contagious epithelioma, see Avian pox
Convulsion 72, 75, 104, 119
Coots
  aspergillosis in 145
  avian botulism in 92–93
  avian cholera in 71–72, 74, 81
  avian salmonellosis in 101
  gizzard worms in 159–160
  inclusion body disease of cranes in 129, 131
  lead poisoning in 177
Cormorant, oil toxicosis in 193
Cornea 210
Coronary band 210
Cosmopolitan species 210
Cracker shell 40
Crane herpes, see Inclusion body disease of cranes
Cranes 206
  avian cholera in 70–71
  avian salmonellosis in 102–103
  avian tuberculosis in 95
  chlamydiosis in 110
  inclusion body disease of 129–134
  lead poisoning in 177
  oil toxicosis in 193
Crow
  avian cholera in 70–72, 75, 81
  avian tuberculosis in 95
Cyst, in muscle 154, 157

Dabbling ducks, see Puddle ducks
Decapitation 59
Deer, dissection of 202–203
Demoiselle crane, inclusion body disease of cranes in 130
Diarrhea 96, 104, 130
Digestive tract, see also specific organs
  impaction of 185
  oil in 192
Disease contingency plan 21, 55–57
Disease control operations
  analysis of 52
  communications during 51, 54
  equipment for 53–54
  follow-up surveillance 52, 54
  initial response phase
    animal relocation 40–45
    carcass collection 23–27, 51, 53
    carcass disposal 27–39, 51, 53
    disinfection 46–49, 53
    personnel for 46, 50
    problem identification 23, 57
    sanitation procedures 53
  outline of 51–52
  planning of
    background biological data 21–23
    identification of needs 21–23, 51
  work areas for 51, 56
Disease outbreak summary 58
Disinfectant 46
  application of 46–50
Disinfection 46–50

Dissection 7–12
  of bird 10–11
  of mammal 10, 202, 203
Diving ducks 213
  aspergillosis in 145
  gizzard worms in 160
  lead poisoning in 176, 189
  nasal leeches in 166
  oil toxicosis in 193
  sarcocystis in 155
Dog, location of carcasses by 27
Domestic animals 213
  avian botulism in 84
  avian tuberculosis in 95–96
  chlamydiosis in 108
  Pasteurella multocida in 70
Doves
  avian salmonellosis in 102–103
  chlamydiosis in 109, 113
  lead poisoning in 177
Drive trap 41
Droopiness 119
Droppings
  blood-stained 72
  fawn-colored or yellow 72
Drowning, death from 87,89
Drowsiness 102
Drugged grain 41
Dry ice
  for euthanasia 61–63
  in shipment of specimen 13–19
Duck disease, see Avian botulism
Duck leeches, see Nasal leeches
Duck plague 2, 41, 49, 76, 117–127
Ducks
  avian botulism in 87, 89
  avian cholera in 70–71
  avian salmonellosis in 101, 102, 104
  avian tuberculosis in 97
  chlamydiosis in 107–108, 113
  dissection of 10–11
  duck plague in 117–127
  gizzard worms in 159
  lead poisoning in 176
  nasal leeches in 166
  oil toxicosis in 193
  sarcocystis in 153, 155
  scientific names of 205
Duck virus enteritis, see Duck plague
DVE, see Duck plague
Eagles
  avian cholera in 70, 81–82
  lead poisoning in 175, 177, 178
Earth-moving equipment 28, 53
East African crowned crane, inclusion body
  disease of cranes in 130, 132
Ecology 210
Ectoparasites 108
Egg specimen 104
Egret
  chlamydiosis in 107, 108, 110, 113
  oil toxicosis in 193
  sarcocystis in 155
Eider, avian cholera in 71, 72, 81
Emaciation 147, 159, 179, 192
Emasculatome 210
Environ 46, 53
Environmental changes 1
Epizootiology 210
Epomidiostomum 159
Escape response, absence of 179, 181
Esophagus
  cheesy raised plaques in 123, 126
  impaction of 179, 185
Etiologic agents 210
  shipping regulations concerning 19–20
European jay, aspergillosis in 145
European sparrowhawk, avian pox in 135
European starling
  avian salmonellosis in 101
  avian tuberculosis in 95
Euthanasia 59–63
  checklist for 63
  chemical 60–63
  physical 59–60
Evening grosbeak, avian salmonellosis in 101
Exsanguination 60
Eyelids
  paralysis of 87, 90
  pasted 102
Eyes
  leeches on 165, 168
  pussy exudate of 107
  wartlike nodules on 137, 138, 140

Face, wartlike nodules on 139
Falcons 205, 206
Fat, loss of subcutaneous 183
Fecal specimen 102
Feces, bright-green 179, 181

Feeding, difficulty in 139
Feeding area 25
Feet, wartlike nodules on 137, 139, 141
Finch, avian pox in 135
Fish and Wildlife Regulations for packaging 19
Fixation 7–10
Flamingos 205
Flies 135
Flight
  erratic 72, 179
  inability to sustain 179
  loss of 5, 86, 90
Formalin 209
Four-wheel vehicle 27, 53
Fowl cholera, see Avian cholera
Fowl pox, see Avian pox
Fowl typhoid, see Avian salmonellosis
Freezing of specimen 10
Frigate, oil toxicosis in 194
Fulmar
  chlamydiosis in 107
  oil toxicosis in 194
Funnel trap 41

Gadwall
  avian cholera in 72
  duck plague in 118
  nasal leeches in 165
  sarcocystis in 153
Gait, unsteady 179
Gallbladder, bright green bile in 179, 187
Gaping 147–148, 165
Gas
  anesthetic 61
  toxic 61–63
Gastrointestinal tract, hemorrhage from 123, 125
Geese
  avian cholera in 70–71
  avian salmonellosis in 101–102
  avian tuberculosis in 97
  chlamydiosis in 107–108
  duck plague in 117, 123
  gizzard worms in 159–160
  lead poisoning in 176, 185
  nasal leeches in 165–166
  oil toxicosis in 193
  sarcocystis in 153, 155
  scientific names of 205
Gizzard 210
  green 179, 187
  hemorrhage of 76, 159–161
  inflammation of 159–161
  sloughing of 159–161
  ulceration of 159–161
  contents of 179, 187–189
Gizzard worms 159–163
  life cycle of 162–163
Gloves 23–27, 53
Goggles 196
Golden eagle
  avian pox in 135
  lead poisoning in 177
Goldeneye
  avian pox in 135
  lead poisoning in 175
Grackle
  avian salmonellosis in 101
  sarcocystis in 153
Great blue heron, oil toxicosis in 195
Grebes 205
  avian botulism in 92–93
  avian cholera in 70
  gizzard worms in 159
  oil toxicosis in 193
Grouse
  avian salmonellosis in 102
  lead poisoning in 177
Guineafowl, avian salmonellosis in 102
Gulls 213
  avian botulism in 83, 85
  avian cholera in 70, 71, 81
  avian salmonellosis in 101–103
  avian tuberculosis in 55
  chlamydiosis in 107, 109, 110
  gizzard worms in 160
  lead poisoning in 177
  oil toxicosis in 193
  sarcocystis in 156

Hatchet-breast appearance 179, 182, 210
Hawaiian forest birds 213
  avian pox in 137
Hawaiian goose, avian pox in 137
Hawks 204–205
  avian cholera in 70, 72
  avian salmonellosis in 102
  lead poisoning in 177
Hazardous Material Regulations 19

Head
  dropping forward 122
  hanging down 5
  inability to hold erect 87, 90
  pulled back and to side 122
  shaking of 165
  swollen 184
  thrown back between wings 72, 75
Heart, hemorrhage on surface of 76–79, 123, 126
Helicopter 27, 40, 53
Hemorrhage
  of gizzard 159–161
  on gizzard surface 76
  in gastrointestinal tract 123, 125, 130, 132
  on heart surface 76–79, 123, 126
  in thymus gland 130
Herons 205
  avian botulism in 85
  avian cholera in 70
  chlamydiosis in 107, 109, 110
  oil toxicosis in 193, 195
  sarcocystis in 155
Herpesvirus 117, 129, 210
Herring gull, aspergillosis in 148
Heterogeneous organism 211
History, see Specimen history
Hooded crane, inclusion body disease of cranes
  in 130, 132
House sparrow
  avian salmonellosis in 101
  avian tuberculosis in 95
Human health risk
  of aspergillosis 150
  of avian botulism 92
  of avian cholera 82
  of avian salmonellosis 106
  of avian tuberculosis 96, 99
  of chlamydiosis 113
  of lead poisoning 189
  of oil spill 196
Hunter-crippled waterfowl 145
Husbandry practices 210
Hypothermia 191

Ibis 205
  chlamydiosis in 110
  sarcocystis in 155
Immunosuppressive therapy 211
Impaction 211
Inapparent infection 211
Incineration of carcasses 27–28, 53
  above-ground 27–28
  in-trench 27–28
  layering of carcasses for 27, 39
Incinerator, portable 27
Inclusion body disease of cranes 129–134
Infectious agent 211
Intermediate host 211
Internal combustion engine, as carbon monoxide
  source 61
Intestine
  hemorrhage in 130, 132
  necrotic bands around 123, 125
  nodular lesions in 97, 98
  raised plaques in 123, 126
  reddened or coated 102
  thickened, yellowish fluid in 76, 80
Island, carcasses along shoreline of 86, 88

Killing chamber 61–63

Labeling of specimen 7–9, 10
  for shipment 19–20
Lameness 96, 153
Landing, poor 179
Latent disease 211
Laysan albatross, avian pox in 135, 137, 139–140
Lead-chelating chemicals 189
Lead poisoning 147, 175–189
Leeches, nasal, see Nasal leeches
Legs
  loss of strength in 5
  wartlike nodules on 137, 139–140
Lethal injection 60–61
Lethargy 72, 75, 96, 130, 132, 211
Light, hypersensitivity to 119
Limberneck, see Avian botulism
Liver
  exudate of 112
  necrotic spots in 123, 126
  nodular lesions in 97, 98
  paratyphoid nodules in 102
  swollen 130, 132
  thickening of 112
  yellow-white spots in 76, 79, 130, 132
Livestock, see Domestic animals
Loons 205
  avian botulism in 83, 85
  aspergillosis in 145
  oil toxicosis in 193
Louisiana pneumonitis, see Chlamydiosis

Lungs
  flattened yellow plaques in 147, 148, 149
  nodular lesions in 97
  oil in 192

Maggots 83–84, 89, 92
Mallard
  aspergillosis in 145, 147
  avian botulism in 93
  avian cholera in 72, 75
  avian pox in 135
  duck plague in 117–120, 125
  lead poisoning in 175, 180, 182, 187
  nasal leeches in 170
  sarcocystis in 153, 157
Mammals
  chlamydiosis in 108
  dissection of 9, 10, 202–203
  sarcocystis in 153
  scientific names of 206–207
Manchurian crane, inclusion body disease of cranes in 130
Maps 21–22, 25, 56
Marine birds 213
  avian pox in 136, 137
Marsh, botulism in 92
Mergansers 213
  lead poisoning in 175, 176
  nasal leeches in 166
  sarcocystis in 155
Migratory birds 213
Mink, avian botulism in 84
Molting birds 87
Mosquitos 135, 141
Mourning dove
  avian pox in 135, 139
  avian salmonellosis in 102
Mouth, mucous discharge from 72
Murres
  avian pox in 135, 139
  oil toxicosis in 191, 194
Muscle
  calcification of 154, 209
  cyst in 154, 157
  loss of 96, 153, 179, 182
Muscovy
  duck plague in 118
  inclusion body disease of cranes in 131
Mute swan
  avian pox in 135, 139
  avian salmonellosis in 101
  lead poisoning in 176
Mycobacteriosis, see Avian tuberculosis
Mycobacterium avium, see Avian tuberculosis
Mycotoxin 211

Nasal discharge
  bloody 72, 119, 121
  pussy 107
Nasal leeches 165–171
National Wildlife Health Center, shipment of specimen to 16–18
Neck
  crooked 179
  paralysis of 87, 90
Neck droop 2
Necrosis 211
Necropsy 211
Nictitating membrane 211
  paralysis of 87, 90
Nodule 211
Nomenclature, scientific 205–207
Nontoxic shot 189
Northern bobwhite, avian pox in 135, 139
Northern cardinal, avian salmonellosis in 101

Occlusion 211
Oil spill 145, 191
  frequency of kinds of 192
  geographic distribution of 194
Oil toxicosis 191–196
Onset of disease 1
Organ specimen, see Tissue sample
Ornamental game birds, avian tuberculosis in 95
Ornithosis, see Chlamydiosis
Owls 206
  avian cholera in 70
  avian salmonellosis in 102
  lead poisoning in 177

Packaging of specimen, see Shipment of specimen
Packing list 19
Parakeets 206
  avian salmonellosis in 102
  chlamydiosis in 107, 108, 113
Paralysis 86, 87, 89, 153
Paratyphoid, see Avian salmonellosis
Parrot disease, see Chlamydiosis
Parrot fever, see Chlamydiosis

Parrots 206
  avian salmonellosis in 102
  avian tuberculosis in 95
  chlamydiosis in 107, 109, 113
Passerine birds
  aspergillosis in 145
  sarcocystis in 153
Pasteurella multocida, see Avian cholera
Pathogenic organism 211
Peafowl, avian salmonellosis in 102
Pelicans 205
  avian cholera in 70
  gizzard worms in 160
  oil toxicosis in 191, 193
Penguins 205
  aspergillosis in 145
  avian salmonellosis in 102
Penis, prolapse of 119, 121
Perching birds 206
  avian pox in 135
Pericardium 211
  exudate on 112
Pesticide poisoning 119
Petrel, oil toxicosis in 194
Pheasants
  avian botulism in 83, 84, 86
  avian cholera in 70, 72
  avian salmonellosis in 102
  avian tuberculosis in 95
  lead poisoning in 177, 189
Photography 1-5
Physical euthanasia 59-60
Pigeons 206
  avian cholera in 70
  avian salmonellosis in 101-102
  chlamydiosis in 107-109
Pine siskin, avian salmonellosis in 101
Pintail
  avian botulism in 83, 84, 86
  avian cholera in 72, 75
  duck plague in 118
  lead poisoning in 183
  sarcocystis in 153
Pit, burning 27
Plague 212
Plastic bag
  for specimen handling 7-9
  for specimen shipment 13, 16-17
Platform, burning 27
Plover, oil toxicosis in 193
Plumage, darkening and dulling of 96
Plumbism, see Lead poisoning
Pneumonia, brooder, see Aspergillosis
Poisoning, see specific kinds of poisoning
Population at risk 4
Population data 24
Population management 54
Population movement 4, 21-25
Poultry 213
  aspergillosis in 145
  avian botulism in 83, 86
  avian cholera in 70
  avian salmonellosis in 101-102, 106
  avian tuberculosis in 95
  chlamydiosis in 107-108, 113
  gizzard worms in 159
  inclusion body disease of cranes in 131
Pox, avian, see Avian pox
Poxvirus infection, see Avian pox
Preservation of specimen 7-12, 169, 171
Propane exploder 40
Protective clothing 23 28
Protoporphyrin IX 182,212
Proventriculus 212
  cheesy raised plaques in 123, 126
  impaction of 179, 185
  contents of 179
Psittacines 212
  avian tuberculosis in 95
Psittacosis, see Chlamydiosis
Public use documents 57
Puddle ducks 213
  avian cholera in 74
  gizzard worms in 160
  lead poisoning in 175-176, 189
  nasal leeches in 166
  sarcocystis in 155
Puffin, oil toxicosis in 191, 194
Pullorum disease, see Avian salmonellosis

Quail
  avian pox in 135
  avian salmonellosis in 102
  avian tuberculosis in 95
  lead poisoning in 177

Rail, lead poisoning in 177
Raingear 23, 26, 46, 53

Raptors 213
  aspergillosis in 145
  avian botulism in 84–85
  avian cholera in 70–71
  avian pox in 135, 137
  avian salmonellosis in 103
  avian tuberculosis in 95
  chlamydiosis in 110
  gizzard worms in 160
  lead poisoning in 177, 189
  oil toxicosis in 194
  sarcocystis in 156
Rectum, enlargement of 102
Red-crowned crane, inclusion body disease of cranes in 131
Redhead
  avian cholera in 72
  duck plague in 118
  lead poisoning in 175
  nasal leeches in 165
Red-shouldered hawk, avian pox in 135
Red-tailed hawk, avian pox in 135
Red-tailed tropicbird, avian pox in 135, 137
Relocation of animals 40–45
Rendering of carcass 27 – 28
Reptiles, sarcocystis in 153
Retriever club 27, 46, 179
Rice breast disease, see Sarcocystis
Ring-necked duck, nasal leeches, in 165
Rock dove, chlamydiosis in 107, 109, 110
Rocket net 41–42
Rodents 208
  avian salmonellosis in 101, 106
  chlamydiosis in 108
  Pasteurella multocida, 70
Ross' goose, lead poisoning in 176
Rough-legged hawk, avian pox in 135
Ruddy duck, nasal leeches in 165
Ruminants 213

Salmonellosis, see Avian salmonellosis
Salt glands, swollen 192
Sandhill crane, inclusion body disease of cranes in 129–131
Sandpiper, oil toxicosis in 193
Sarcocystis 153–158
  life cycle of 158
Sarcocystosis, see Sarcocystis
Sarcosporidiosis, see Sarcocystis
Sarus crane, inclusion body disease of cranes in 130, 132
Scaup
  aspergillosis in 145
  avian cholera in 72
  lead poisoning in 175
  nasal leeches in 165
Scavengers 213
  avian tuberculosis in 95
Scientific names 205–207
Scoter
  avian cholera in 74
  avian pox in 135
  nasal leeches in 165
Scrubber filtration tank 61–62
Sea ducks 213
  avian tuberculosis in 97
  gizzard worms in 160
  lead poisoning in 176
  nasal leeches in 166
  oil toxicosis in 193
  sarcocystis in 155
Section 7 consultation 212
Shearwater, oil toxicosis in 194
Shipment of specimen 13 – 20, 200 – 201
  by commercial carrier 20
  federal regulations concerning 19
  label required for 19–20
  to National Wildlife Health Center 16
  preventing breakage and leakage during 13
  refrigerated 13
  supplies for 14
Shooting (physical euthanasia) 60
Shore, carcasses along 86, 88
Shorebirds 214
  avian botulism in 84, 85, 89, 92
  avian cholera in 71
  avian pox in 136
  avian salmonellosis in 103
  chlamydiosis in 107, 109
  gizzard worms in 160
  lead poisoning in 177, 189
  sarcocystis in 156
Shoveler
  avian botulism in 93
  lead poisoning in 176
  nasal leeches in 165
  sarcocystis in 153
Snow goose
  avian cholera in 72, 74, 82
  chlamydiosis in 113
  gizzard worms in 159, 162
  lead poisoning in 175, 176, 180
  sarcocystis in 153

Songbirds 214
  avian botulism in 85, 86
  avian cholera in 70–71
  avian pox in 136–137
  avian salmonellosis in 102–103
  chlamydiosis in 109–110
  oil toxicosis in 194
Specimen, see also Carcass; Tissue sample
  choice of 7
  collection of 7–12, 200–201
  containers for 13–15
  dissection of, see Dissection
  labeling of 7–9, 10
  preservation of 7–12, 200–201
  shipment of, see Shipment of specimen
  shipping of 200–201
Specimen history 1–5
  form for 197
Spleen
  nodular lesions in 97, 98
  swollen 130, 132
  yellow-white spots in 130, 132
Spoonbills 205
Staggering 102
Stanley crane, inclusion body disease of cranes
  in 130
Stomach worms, see Gizzard worms
Stunning 60
Styrofoam cooler 13–19, 201
Swans
  avian botulism in 87
  avian cholera in 70
  avian salmonellosis in 102
  duck plague in 117, 123
  gizzard worms in 159–160
  lead poisoning in 176
  nasal leeches in 166
  oil toxicosis in 193
  sarcocystis in 153, 155
  scientific names of 205
Swimming, circular 72, 75, 122

Tail, fanned and rigid 122
TB, see Avian tuberculosis
Teal
  avian botulism in 93
  avian cholera in 72
  avian pox in 135
  duck plague in 118
  lead poisoning in 176
  nasal leeches in 165
  sarcocystis in 153
Tern
  avian cholera in 81
  chlamydiosis in 110
  gizzard worms in 160
  sarcocystis in 156
Theromyzon 165
Thirst 119
Thymus gland 212
  hemorrhage in 130, 132
Ticks 108
Tissue sample
  collection of 7–12
  containers for 7–9
  fixation of 7–9
  labeling of 10
  preservation of 7–12
Toxic gas 61–63
Toxicosis, oil, see Oil toxicosis
Trachea, oil in 192
Trembling 102
Trumpeter swan, nasal leeches in 165, 169, 170
Tuberculosis, see Avian tuberculosis
Tumbling 102
Tundra swan
  aspergillosis in 145
  avian pox in 135
  lead poisoning in 175, 176, 181, 185
  nasal leeches in 165

Ulcers 123
Unthrifty appearance 96, 102, 147, 212
Upland game birds 214
  avian cholera in 70–71
  avian pox in 136–137
  chlamydiosis in 110
  lead poisoning in 177

Vaccine
  against avian cholera 82
  against avian pox 141
  against duck plague 127
Vent
  bloody discharge from 119, 121
  oil in 192
  pasted 102
  stained bright green 179, 184
Ventricular nematodiasis, see Gizzard worms
Vultures 205–206
  avian botulism in 84

Wading birds 214
  avian cholera in 71
  avian pox in 136
  avian salmonellosis in 103
  gizzard worms in 160
  oil toxicosis in 193
  sarcocystis in 156
Wastewater discharge 92, 99, 104, 191
Watercraft 27, 29-30, 46, 53
Waterfowl, see also Ducks; Geese; Swans 205, 214
  aspergillosis in 145
  avian botulism in 84, 85, 86, 87, 88, 91, 92
  avian cholera in 69
  avian pox in 135-136
  avian salmonellosis in 103
  avian tuberculosis in 95
  capture devices for 41
  chlamydiosis in 107-110
  hunter-crippled 145
  lead poisoning in 175, 178, 179, 181, 189
  nasal leeches in 165
  population data on 23, 56
  sarcocystis in 153
Weakness 102, 107, 153, 159
Western duck sickness, see Avian botulism

Whirl-Pak bag 7-10, 17
Whistling duck, lead poisoning in 176
White-fronted goose, avian cholera in 72
White-naped crane, inclusion body disease of cranes in 130
White Pekin duck
  duck plague in 117, 127
  inclusion body disease of cranes in 129, 131
White-tailed tropicbird, avian pox in 139
White-winged dove, chlamydiosis in 113
Whooping crane, avian cholera in 81
Wigeon
  avian cholera in 72
  nasal leeches in 165
  sarcocystis in 153
Wildlife disease programs (U.S.) 199
Wing droop 2, 102, 147-149, 179, 181
Wings
  extended from sides 122
  in roof-shaped position 179-180
Woodcock, lead poisoning in 177
Wood duck
  aspergillosis in 145, 147, 150
  duck plague in 118
  lead poisoning in 176
Worms, gizzard, see Gizzard worms

Friend, Milton, editor. 1987. **Field guide to wildlife diseases.** U.S. Fish Wildl. Serv., Resour. Publ. 167. 225 pp.

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